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THE
LARYNGOSCOPE.

VOL. LIX DECEMBER, 1949. No. 12

PSYCHOSOMATIC APHONIA AND EPHEMERAL ADDUCTOR PARALYSIS.

CHEVALIER JACKSON, M.D.,
Philadelphia, Pa.

Psychosomatic laryngeal manifestations usually affect the most conspicuous function; namely, phonation. Of the other eight functions, the tussive is practically the only one thus affected. As shown by Jackson and Jackson,[1] the old adjectives, hysterical, voluntary, functional, formerly misapplied to this condition are semantic solecisms and should be dropped from the literature.

Etiology and Pathology: As stated by the just mentioned authors, most of the cases are essentially traumatic neuroses. They found that many cases originated in *ephemeral adductor paralysis.*[1] This transitory paralysis, they point out, is due to sudden circulatory disturbance, usually anemic, of the anterior cerebral cortex. In common parlance this condition is often referred to as "knocked speechless," "speech bound," "speech bereft," "dumbfounded," "struck dumb." Though these expressions are common in lay literature, their significance does not seem to have been recognized by medical authors other than those cited.[1] The sudden anemia of the cerebral cortex in the minor form of syncope called fainting or swooning is

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commonly associated with an ephemeral adductor paralysis. This phase, in swooning, passes so quickly into unconsciousness that the adductor paralysis is not ordinarily noticed, but the authors mentioned have observed it in the laryngeal mirror in cases of fainting from various causes.[1] When the degree of cerebral anemia is not quite sufficient to cause loss of consciousness, the person affected by a terrific sight or a shocking experience notices his loss of power to speak, hence his expression, "I was struck dumb," or "I was dumbfounded," or "I was knocked speechless." In many instances of fainting or swooning, the person affected, after recovering consciousness, will recall the loss of power to speak.[1]

Pathologically, the laryngeal condition of failure in adduction in ephemeral adductor paralysis is, in a sense, somal rather than functional, inasmuch as the cells of the anterior motor cortex are deprived of their blood supply. When, however, the psychic impression of inability to speak becomes fixed, as is sometimes the case, a phychosomatic aphonia is established. This is only one of the ways in which this type of aphonia originates. Various forms of suggestion may become fixed. In some cases anxiety over prospect of loss of voice, suggested by acute laryngitis or other incident, may be an etiologic factor. These patients are usually occupational voice users. In most instances psychosomatic aphonia is a traumatic neurosis. Physical trauma may or may not have been concurrent, but its effect is through its additional mental impact. In some cases the suggestive physical trauma may have been in the region of the larynx.[1]

Psychosomatic aphonia may develop immediately after the psychic trauma or later. The onset is usually sudden, but in some cases it is gradual. When predisposing factors result from overwork, grief, anxiety, worry and other long continued strains, the memory of any incident referable to the voice, or even the larynx, may result graually in a psychosomatic aphonia, though often even in these cases the onset is sudden. In some cases the etiologic suggestion may come from loss of voice affecting a relative or even from a vivid account of such a loss.

Though in some cases predisposing factors cannot be traced, yet in most cases such factors are found. As with other psychosomatic conditions, hereditary factors may contribute to vulnerability under strain. There is a wide variation in powers of resistance. We are not all born with the same amount of resistance to the ill effects of the anxieties and worries of life. Many are born of parents whose hereditary backgrounds contain cases of psychopathic or psychotic disorders. Excesses, especially alcoholic, on the part of parents tranmit deficiencies in powers of resistance to the ill effects of strain. This factor is progressively on the increase.

Symptoms: The chief complaint of the patient is that he has lost his voice. By this he may mean that he has lost only his phonatory voice or both phonatory and whispered voice. In the latter case, which is less common, the patient may write everything he has to say or he may make efforts to communicate by signs. He may just listen attentively to the description of his disability by an accompanying relative, friend or physician. When asked a question he may simply shake his head or he may make prodigious movements of his facial muscles without uttering a laryngeal sound. If he has not lost his ability to whisper he may whisper freely or be brief and reticent. He may seem distressed about his disability or proud of it, as if it were interesting in its rariety and mystification.[1]

Laryngeal Appearances: To the laryngologist who is accustomed to seeing the approximation of the vocal cords when the patient attempts to say E-e-e-e with the mirror in place, three significant abnormalities in a typical case of psychosomatic aphonia are striking: *a.* The vocal cords remain in the quiet breathing position; *b.* the arytenoid eminences are motionless; *c.* the larynx fails to rise. With this clear picture of the typical case in mind, the slight variations in laryngeal appearance will be easily detected. In some cases at times the cords will be seen to go part way toward the midline, then fall back outward again to the quiet breathing position. If asked to say E-e-e-e again, the partial excursion may or may not be repeated. When the patient is asked to cough, the vocal cords are seen to approximate for a moment, then permitting

the expulsion of a puff of air carrying with it the sound waves characteristic of cough. The sound of the cough is the normal one of glottic closure. As a matter of differential diagnosis it is important to remember that if the patient should cough without glottic closure the case is not one of psychosomatic aphonia. It will be noticed that the cough without glottic closure has a harsh rasping sound of air waste.

Diagnosis: The mirror appearances are diagnostic unless the patient refuses to cough. In this case direct laryngoscopy should be done without anesthesia. At the approach of the direct laryngoscope the glottis will be snapped shut by the adductor muscles. One important diagnostic sign, unmentioned in the literature, is the sound of glottic cooperation in cough. The sounds of presence and of absence, respectively, are unmistakable to the practiced ear (Case 5). Every human being has to cough occasionally whether he wants to do so or not; and it can be provoked my various means. This sign of approximative power, of course, has its greatest usefulness in cure of uncooperative patients. With approximative power present, willful silence remains to be excluded. This diagnostic possibility may be in one of two forms, psychotic and purposeful. As a feature of a psychosis it occurs as the willful stubborn silence called aphonia paranoica. The features of a psychosis are so conspicuous that diagnosis presents little difficulty. The diagnosis of the silence of malingering is not always so easy. The malingerer knows he can talk, but refuses to do so for a definite purpose, such as to escape duty or to perpetrate a fraud. Claims for disability may be on the basis of a loss of voice alone or in combination with simulation of other forms of disability, or, oftener, in combination with well substantiated physical injuries. Compensation claims are usually for relatively small amounts; but claims for damages may be large in case of occupational voice users and enormous in case of professional vocalists. This matter of purpose is important diagnostically, and should be searched for if not evident. In a number of cases the patient was brought in for consultation by the family physician from whom the ulterior motive of a damage suit had been concealed. There is one

important sign of malingering to be noted by careful attention to the manner of the patient. He is suspicious of a trap that will reveal his fraudulent purpose If carefully watched for, there will be noted a manner of hesitation and suspicion preceding his compliance with requests. He does not freely and frankly cooperate in examinations and tests. He may not consent; if he does, it is likely to be partially or delayed. When these signs are noted psychosis must be eliminated, as suspicion is often present in psychopaths and in psychotics. Having eliminated the possibilities, the probability of malingering is put to the final test, which is therapeutic. Phonation can be restored by treatment if there is no psychotic trouble and no malingering. If the methods of the laryngologist fail to restore the voice, the patient should be turned over to the psychiatrist for treatment. The malingerer will usually balk at this.

Treatment: The first question to be decided is as to when treatment should begin. Broadly speaking, there are two classes of cases to be considered. In the first class are the cases in which the chief and determining cause has been a violent and shocking episode occurring when the patient was in relatively good health. In the second class are the cases in which the aphonia has been largely due to prolonged strain and nervous exhaustion. In the first class of case the treatment should be begun as soon as the novelty of the situation has worn off and the patient has or can quickly develop a desire to get the voice back. In the second class of case a period of rest under general medical care and management is essential. It is a serious mistake to begin treatment while the patient is fatigued, exhausted and depressed. There are a number of reasons: *1.* These conditions are causes, hence should not be allowed to continue. *2.* The patient, worn out and in need of rest, really cares little whether he gets well or not; hence it is difficult or impossible to create the essential confidence and cooperation. *3.* Failure is almost certain. *4.* Failure enormously increases the difficulty of subsequent cure, because *a.* it discourages the patient; *b.* it renders him skeptical as to cure; *c.* he is almost certain to recognize simi-

larity of technique of treatment in which he has lost faith; *d.* it is always best, if possible, to carry through the psychic treatment to successful phonation at the first seance.

The basic condition in psychosomatic aphonia being essentially psychic, the treatment of it is essentially psychic. This form of treatment for success requires certain personal qualifications that many physicians do not possess. The neurologist and the psychiatrist, of necessity, have these qualifications well developed. The personal qualifications required in psychosomatic aphonia cannot be accurately described. The manner must be firm, self-confident, never discouraged or taken back, never admitting failure. Though dominating, his manner must be kindly, never irritated or annoyed. Certain errors must be avoided. The patient must not be told that he could talk if he wanted to. He must not be allowed to think his ailment is mysterious, puzzling or problematical.

For success it is well for the physician to get the conception that the abnormality in psychosomatic aphonia is that the patient *believes* he has lost his voice. It is not imaginative in the ordinary sense of that term. It is not willful nor intentional. It is a belief rather than an illusion, delusion or hallucination.

The first step is to inspire confidence in the patient. He should be given a simple statement such as that his trouble is a well known form of imperfect coordination of vocal muscles, and is curable by a system of exercises.

The next step is to get the patient to make a sound in some way other than an effort to say a word. The method mentioned in the case reports is usually successful and illustrates what is meant by a method other than saying a word. To try to say a word meets and rarely surmounts the patient's basic difficulty. Producing a sound in some other way can be made to impress the patient that he is getting a start on learning to talk by a method that is free from his difficulties. When the patient produces a phonatory grunt by bringing his elbows down with a thump against his sides he can be easily convinced that he has started learning a new way of talking.

Further steps of a successful plan of treatment will be given in connection with reports of cases in which it was used. The report of a typical case of each of the most frequently encountered varieties of psychosomatic aphonia will serve to illustrate the most important clinical features better than a space consuming large series of case reports which would be confusing and perhaps unread.

Case 1: A woman, aged 22, supported her ill and widowed mother by working as a telephone operator in a hospital. Long hours of duty were followed by long hours in care of her mother and their two rooms. While she was busy at her switchboard, a psychotic patient came in and garroted her to unconsciousness before he could be forced by rescuers to release the grip of his hands on her throat. She was quickly resuscitated. Examination in the surgical department was negative except for a slight swelling of the soft tissues of the neck and a few ecchymotic spots on the skin. There was no dyspnea, and Roentgen examination showed no evidence of fracture of the hyoid bone or thyroid cartilage. On the tenth day after the garroting, the patient was still in the hospital, and laryngeal examination was requested because of loss of voice. The patient was found crying and, when asked why, she wrote, "I have lost my voice and will lose my position as a telephone operator. What will my poor mother do?" The night nurse reported the patient was restless, cried frequently through the night and moaned, though she did not say a word. Palpation of the neck and careful palpation of the laryngeal cartilages revealed no tenderness or other laryngeal abnormality. Mirror examination showed the glottis in the position of quiet breathing. When the patient was asked to take a deep breath, the cords increased their separation normally. When the patient was asked to say "E-e-e-e," she shook her head slightly, and no movement of the glottic margins was perceptible in the mirror. When asked to say "Ah" they were motionless. When asked to cough, the patient made an effort to do so; the cords promptly and normally approximated for a moment and the characteristic sound of cough with glottic cooperation was plainly audible. Obviously the aphonia was psychosomatic. The patient was told that shock had caused a little imperfect muscular action in the larynx; that she would get well under treatment, and that she would have a good voice for continuing her work as a telephone operator. She was assured that this would be done by a method that amounted to learning to talk in a new way. Her confidence and hearty cooperation were obtained. The start in phonation was made by the patient bringing her bent elbows forcibly to her sides in a thump coincident with a grunt. Her attention was then called to the fact that the sound of the grunt was the start, and that the next step would be the vowel sounds. The first was "O." She watched the physician's mouth as he emitted a clear, strong "Oh" sound. She promptly sounded the vowel. Then the other vowels followed in succession. She was then enthusiastic and talking freely; and did not need to be trained on consonants. In two weeks she was back on duty at the switchboard and there was no recurrence.

Comment: The fright and subjective sense of imminent death by suffocation produced a profound shock. When she became conscious her vivid recollection of those hands pain-

fully gripping her neck fixed her attention on her larynx. Her first attempts to speak were naturally feeble and imperfect, she assumed permanent injury had been done to her larynx. Her anxiety about her mother, the prospect of losing her voice and the means of support for herself and her invalid and widowed mother, all were typical causes of psychosomatic aphonia. Her long hours of overwork at the switchboard and at home were predisposing factors. Few cases are so complete. Only an hereditary background suggesting vulnerability was lacking. Direct violence in the region of the larynx is uncommon; though we have seen a few cases, of which the following is another example:

Case 2: A front brakeman on a freight train, aged 28, was referred for an opinion because of a claim for permanent disability based on total loss of voice. The history was that the loss of voice followed an injury by a blow on the side of the neck while on duty in the cab of a locomotive. The fireman was pulling clinkers when a sudden coming away of a clinker allowed the proximal end of the clinker hook in the fireman's clenched fist to strike the claimant on the side of the neck. The force of the blow knocked him over onto the loose coal in the tender, which caused him to lose his footing and fall. In falling he slid almost off the train between the cab and the tank, his head, shoulders and arms dangling out. When dragged back to safety, he tried to curse the fireman for carelessness but found himself speechless. The company surgeon found no evidence of cervical trauma, but the inability to phonate had persisted for two months when an attorney strongely urged putting in a disability claim. A consultant for the defense thought malingering was indicated by the fact that the whisper was also gone. The patient was brought for an opinion on the diagnostic problem. Mirror examination showed the normal image of quiet breathing with symmetric separation of the cords as the base of the glottic triangle lengthened in response to a request that a deep breath be taken. When asked to say "E-e-e-e" the patient shook his head, and no movement of the cords or arytenoids was perceptible in the mirror. When the patient was asked to cough, the cords were seen to approximate for a moment and the typical sound of cough with glottic cooperation was audible. Obviously a diagnosis of psychosomatic aphonia required only elimination of malingering for the purpose of supporting a disability claim as alleged by the defense. The claimant gave the impression of sincerity. The family physician stated the claimant had been reluctant to sign claim papers. During the laryngeal examination claimant had cooperated promptly and willingly in every way, with no hesitation that would imply suspicion of a trap. He was perfectly willing to have Roentgen examination and laboratory tests made. These evidences of sincerity called for a therapeutic test, thorough and repeated a number of times if negative. The hearty cooperation of the patient in the same plan of treatment as in the preceding case resulted in restoration of the voice and in the dropping of the claim for disability.

Comment: The etiologic factor of direct trauma inflicted in the laryngeal region, though undoubtedly important in both

this and the preceding case, is relatively rare. The trauma is usually only psychic, as in the following case:

Case 3: A woman, aged 23, saw an automobile backing out of the garage toward her two-year-old child who was standing in the neighbor's driveway. She ran and waved her arms, then fell in syncope as she saw the child's head crushed under the rear wheel. When resuscitated she tried to speak but evidently could not utter a word. The next day she was able to whisper, but she could not phonate. She stated that she had tried to scream to warn the driver, but she could not utter a word. The horror at the sight of her child's head being crushed and the speechless condition that rendered her powerless to scream a warning to the driver were the last two things she remembered. Mirror examination of the larynx showed no phonative approximation when the patient was asked to say "E-e-e-e," but good phonative approximation on coughing. Oviously the case was one of psychosomatic aphonia. In consultation with the family physician it was decided best to postpone treatment of the aphonia until proper care had been given to the patient's grief stricken state and profound shock. About a month later there was great improvement in the general condition of the patient, but the voice had not returned and mirror examination showed no change. Treatment of the plan mentioned above was successful in restoration of the voice at the first seance.

Comment: In this case there was no hereditary factor found in the history. The patient was not neurotic; there had apparently been no particular strain prior to the horrible spectacle. There was no direct physical trauma. All the features of the case justified the opinion that the chief cause was the psychic trauma that produced the ephemeral adductor paralysis. The fixation on the phonatory function was due to the powerful mental impression of loss of voice at the moment she needed it in her effort to prevent her child from being crushed to death.

Case 4: A veteran of World War II, aged 29, brought with him a lengthy self-written statement of his case, from which the significant points are here abstracted. After nearly three months of active service at or near the front, he was evacuated to a base hospital as a case of battle fatigue. His chief complaint at that time was loss of voice. He was unable even to whisper. He could whistle to call attention, and he made signs when necessary to call attention to his needs. He said he lost his voice suddenly just as he had almost recovered from hoarseness accompanying an attack of the then prevalent acute laryngitis. The whisper he used during the attack of laryngitis to save his larynx was lost at the same time. At the base hospital, under a regimen of rest, his voice did not return, but his general condition improved to a point at which his request to be returned to active duty was granted. He was assigned to light duty on a hospital ship. Two months later, his term of enlistment having expired, his request for release from service was granted by honorable discharge. His father, a physician, brought him in for consultation concerning his loss of voice, his only remaining disability. Mirror examination revealed no laryngeal abnormality except that there was no perceptible approximation of the vocal cords, nor any

movement of the arytenoids, when the patient was requested to say "E-e-e-e." He seemed to make a sincere effort to phonate, but the only response to the effort was a movement of some of the facial and cervical muscles. When asked to cough, these muscles did not seem to move, but prompt though momentary approximation was noted in the mirror. In beginning treatment, enthusiastic cooperation of the patient was obtained when he became convinced that his phonatory cough and the thumping grunt constituted a good beginning on a series of exercises that would enable him to learn to talk in a new way. He responded well to the vowel exercises. A plainly audible "O" was followed by a louder "A" and by each of the vowels in succession. The first consonant was mastered when, by giving the patient a tooth brush bristle and telling him to place it on the tip of his tongue, hold it against his front teeth, then force it out with a blast of air in the usual way. He was familiar with the technique from personal experience, but he was given an example to imitate. After he had performed the exercise three times he was instructed to add the "O" vowel sound to the dental consonant of the bristle exercise. He was greatly surprised and delighted when he uttered a loud and clear "Toe." The patient and his father carried on the exercises, and the father reported a month later that there was perfect restoration of voice. Three years later the patient was reported free from recurrence.

Comment: Undoubtedly the basic factor in this case of psychosomatic aphonia was the long continued strain, the exhausting daily physical fatigue, along with the impact of almost continuous day and night roar of artillery fire and bomb explosions that rendered any real rest period impossible. Of course, this is well understood in the services, and intervals of rest of personnel are routine; but under active conditions at the front, such intervals for every individual are not always possible, and so it had been in his case. The point germane to our subject is that the prompt response to psychic treatment was due to the long rest in the base hospital and on light duty in the quiet of the hospital ship. A rather large experience with veterans, civilians employed by the services as well as with civilians in private life justifies the conclusion that had the psychic treatment been attempted in the earlier stages of the disorder, failure would have resulted, and, worse yet, the failure would have rendered subsequent cure exceedingly difficult. The patient would have been discouraged, skeptical of help, and would have been likely to detect the technique of treatment and to lack faith in any subsequent effort to help him.

Case 5: A 13-year-old neurotic and atypical boy, whose father died of alcoholism, was recovering from a prolonged manifestation of barking cough of puberty, when he was reproved by his mother for continuous misconduct reported by his school teacher. The next morning he whispered in his mother's ear that his voice was gone and there was no use

going to school because he could not recite. Then he became sullenly silent, refusing to answer questions. He was brought in by the family physician for an opinion as to whether or not there was any laryngeal abnormality concerned in the loss of phonation. The boy was present under protest and promptly, in a hissing whisper, expressed his determination not to allow any kind of examination. Then he was sullen and silent; however, his attitude was moderated by engaging him in an expression of his views of events in baseball. When the conversation lagged he was casually asked if his recent coughing attacks had interfered with his own expert batting. Immediately he emitted a loud barking cough that was unquestionably phonatory and had the characteristic sound of glottic cooperation. In the subsequent report to the family physician it was stated that though this audible evidence of cordal approximation was incontrovertible, yet inferential diagnoses of laryngeal conditions in children should always be subject to direct laryngeal examination. It so happened in this case that removal of badly diseased tonsils had been previously planned, and it was arranged for the rhinolaryngologist to make a direct laryngoscopic examination incidental to the aspiration of blood after the tonsils were out. The rhinolaryngologist found the larynx normal in every way, and reported further that, as the child was coming out of the anesthesia, there was an active glottic closure reflex at the approach of the laryngoscope. The tonsillectomy had no effect on the aphonia. In consultation with the family physician it was decided to advise placing the child under the care of a psychiatrist. Ultimately this patient suddenly developed dementia precox.

Comment: This case is one of a type for which the laryngologist should always be on the alert. The aphonia, though superficially similar to the psychosomatic types, was really a distance signal of the approaching schizophrenia. The vocal phase was a typical aphonia paranoica and not strictly speaking a psychosomatic aphonia. It is advisable for the laryngologist in cases of aphonia to be always alert to psychotic possibilities. Even in mild psychopathic patients there are problems that can be properly dealt with only by a psychiatrist. The psychosomatic aphonia in many psychopaths is only a small part of the problem. The laryngologist's results are usually disappointing in such cases, whereas, under the care of a psychiatrist, except in cases of incurable dementia or of the willful aphonias such as aphonia paranoica, the results are usually good.

SUMMARY.

The most common cause of psychosomatic aphonia is psychic trauma. It often originates in an *ephemeral adductor paralysis* due to sudden anemia of the anterior cerebral cortex as in syncope, commonly called fainting or swooning. In com-

mon parlance this is often referred to as "knocked speechless," "dumbfounded," "struck dumb." Though these expressions are common in lay literature, their significance does not seem to have been recognized by medical authors other than those cited. Etiology, pathology, laryngoscopic appearances, symptoms, diagnosis and treatment are considered and illustrative cases are cited.

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3401 North Broad Street.

II PANAMERICAN CONGRESS OF OTORHINOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

The second PanAmerican Congress of Otorhinolaryngology and Bronchoesophagology will be held in Montevideo-Buenos Aires, Jan. 8 to 15, 1950. For further information communicate with Dr. Chevalier L. Jackson, 3401 North Broad Street, Philadelphia 40, Pa.

THE FENESTRATION OPERATION FOR OTOSCLEROSIS.

A Survey of Experience with 75 Cases Together with a Study of the Effect of the Contralateral Ear.*†

DE GRAAF WOODMAN, M.D.,
New York, N. Y.

To Bárány,[1] in the year 1910, goes the credit for being the first to advocate and perform an opening in one of the semicircular canals for the relief of otosclerosis. Prior to his work, others had tried such procedures as the removal of the stapes by Kessel[2], in 1876, and later, in 1897, Passow[3] made a fistula in the promontory.

It was Jenkins,[4] in 1914, who first made a fistula in the horizontal canal and covered it with a Thiersch graft and then with a flap from the external meatus. Holmgren[5] followed, in 1917, with many years of experimentation. He tried the placing of various substances in the fistulae in an effort to prevent their closure.

Sourdille,[6] in his report of 1935, reported a series of 109 cases with many patients showing good results. His successes resulted from the use of a viable flap made in a three-stage operation which was called the tympanolabyrinthopexie.

The real impetus was given to the surgical approach for the relief of otosclerosis by Lempert[7] in his report of 1938. In this report he described a one-stage operation in which he

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†Presented as Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, Inc.

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used a skin flap like Sourdille's[6] to cover the fistula made from the lining membrane of the external auditory canal. He was able to show a large percentage of cases with lasting improvement proven by audiograms before and after operation. This technique was placed on an even firmer basis when, in 1941, Lempert[8] published a further modification in which he placed the fistula at the ampulated end of the horizontal canal. This he termed the fenestra novovalis. This modification resulted in an even greater number of cases which showed lasting improvement and less tendency to develop bony closure.

This survey of experiences herein reported covers 75 fenestration operations done during the past three and one-half years. The experiences cover complications, accidents, the effect of infection on postoperative results, and an autopsy report with histological findings.

The criteria used for selecting cases for operation was that of a progressive bilateral conductive type of hearing loss in which the air conduction for the critical speech frequencies was below the 30 db level and in which the cochlear function, as indicated by bone conduction, remained above the 30 db level and preferably not below the 20 db level.

Few exceptions were made to this criteria for case selection. There were three cases of unilateral conductive deafness with a family history of otosclerosis. There was one case in which the bone conduction for the 2048 frequency was down to 35 db.

The poorer of the two ears was generally chosen unless one ear had a great deal more tinnitus than the other, in which event the one with the greater amount of tinnitus was selected.

All cases had intact eardrums, patent Eustachian tubes and no evidence of recent ear infection.

ANESTHESIA.

All of these cases were done under general anesthesia with intravenous sodium pentothal together with nitrous oxide and oxygen via an intratracheal airway.

METHOD.

The method used in this series followed the general pattern described by Lempert,[8] namely, the endaural approach with the fenestra made in what he calls the "surgical dome" of the vestibule or ampulated end of the bony horizintal semicircular canal. The fistula is covered by a flap made from the lining membrane of the posterior superior part of the external canal wall continuous with Schrapnell's membrane.

Some modifications have gradually superseded the originally described technique. The triangle incision with removal of skin flap was given up in favor of a single incision, thus removing no skin and leaving a larger flap for filling in the cavity. This is somewhat similar to the technique described by Whitaker, Juers and Shambaugh.[9] Occasionally this flap was made to retract better by two or three short right angle incisions to the edge of the original incision which permitted the flap to fold back more readily into the cavity. The mastoid cavity was only exenterated sufficiently to permit good exposure and working room for the drill to have free action in making the fenestra.

The horizontal canal was always well skeletonized and the facial ridge demonstrated and its mucosal covering elevated forward. Efforts were made to obtain a surface smoothed down well around the canal in order to prevent a deep trough-like exposure of the membranous canal.

The fenestra was made with No. 3 size and No. 2 size dental polishing burrs and for the past two years efforts were made to emphasize the blue line either side of the membranous canal, rather than directly over its midline axis. This, I believe, is similar to the technique described by House[10] and called by him "the double blue line technique."

This technique permits getting off that part of the endosteum directly over the labyrinth, in one large piece, and may thus help in minimizing damage to the endosteal membrane and the membranous labyrinth. Certainly it permits getting most of the fragments and dust away before removing the endosteal membrane. At this writing a recent article by Lempert[11] describes in detail a new technique for removing the endosteal bony layer in one piece, thus avoiding the risk of bony fragments being lost in the perilymphatic space.

For the past 18 months the continuous flow suction apparatus devised by Shambaugh[12] has been used at the time of making the fenestra. We have continued to use loupe magnification rather than the dissecting microscope with its restrictions of free motion.

The flap has always been prepared and thinned out before starting the fenestra. The flap is tucked forward into the external meatus and 1 cc. of 1/1000 adrenalin is poured into the cavity just before starting the fenestra. We have not felt it safe to cover the flap with any protective material such as vaseline gauze or rubber tissue due to lack of room and the fear of catching it in the burr.

In this series two cases were done successfully by the postauricular approach. In three cases a cartilaginous stopple was placed. All three were failures. One of these had a revision and, while no preoperative hearing level was lost, very little, if any, improvement was shown following the revision.

The flap was held in place with a small butterfly made of mesh paraffin gauze placed just above but not directly over the fenestra. A small pledget of cotton covered with sulfathiazol ointment covers the drum. The remaining cavity is packed with wide mesh paraffinized gauze. In some cases melted paraffin was used, but for the past two years this has been replaced with mesh gauze and foam sponge packing, the latter being easier to remove and it also offers less danger of overheating the tissues by the use of too hot paraffin.

POSTOPERATIVE CARE.

Patients were placed on sulfadiazine therapy immediately after operation until two years ago, and since then intramuscular penicillin was used exclusively. At the present time penicillin is given in doses of 250,000 units bd and is administered during the entire postoperative stay in the hospital, which averages 12 days.

Patients are given an infusion during or immediately after operation. Most patients take their fluids the following morning after operation. Rarely is it necessary to carry on infusions after the first 24 hours. Patients are allowed up as soon as the acute dizziness has subsided, which is usually the third or fourth postoperative day.

The outer bandage is generally changed on the second postoperative day. The wound is first dressed on the fifth day. Rarely is the dressing changed on the fourth day, and then only if the patient complains of discomfort, or any elevation of temperature or symptoms which suggest the possibility of a labyrinthitis. At the time of the first dressing all of the packing is removed. A small pledget of cotton is placed in the vestibule of the ear and a vaseline gauze pack is placed between the upper edges of the wound, which helps to prevent this part of the incision from growing together too rapidly. This permits a better exposure and prevents the contracture of the vestibule of the canal, which may cause considerable difficulty in postoperative care.

Our experience has not been as favorable as that reported by some operators on the rapidity of the complete healing and epithelization of the cavity. We have had some ear cavities completely dry in six weeks, but very few. The average is much nearer 12 weeks. One of our best results took over 18 months before all granulations were gone and complete epithelization occurred. All cases have eventually dried up completely.

Audiometric tests are seldom done before the third or fourth week or until the cavity is free of all dressings and active drainage. Voice and tuning fork tests are resorted to during this period.

Routine audiometrics are done 3, 6, 9, 12, 18, 24 and 36 months after operation. Both ears are tested for both air and bone conduction. Masking is done whenever indicated.

COMPLICATIONS

Considering the fact that the fenestration operation is one which requires rather rigid adherence to the technical details of proven step by step procedure, it is surprising that the number of complications are not more frequent than we have experienced.

There were three cases of transient facial paralysis. All of these cases developed this paralysis two to five days after the operation and all cleared up completely within a month of the onset of this complication.

There were two cases of perichondritis. Both occurred three weeks following operation and both were readmitted to the hospital, where through and through incisions were made in the auricle and drains placed. One case was proven to be secondary to a pyocyanus infection and this case was kept covered with dressing soaked with acetic acid, the other case was treated with streptomycin and it in turn improved rapidly under this therapy.

Both these cases had some residual thickening of the auricle but no gross deformity, and the canal remained open and easy to treat. One of the two cases continued on as a good result with complete rehabilitation (method of classifying results to be elaborated upon later in the paper). The other case did not improve but showed no deterioration as the result of this complication.

There was one case of foreign body in the bony labyrinth. A small piece, one-sixteenth inch, broke off the dental excavator and fell into the bony labyrinth, deep to and partially

under the membranous labyrinth. This was recovered by placing the small flap elevator just at the edge of the fistula and then attaching the portable electric magnet used by ophthalmologists. The piece was recovered without damage to the membranous labyrinth, and this case continued on to become one of the rehabilitated cases.

In two cases, perforations of the drum occurred at operation. One of these cases sloughed off the flap in the region of the fistula and continued to show progressive loss of initial improvement; the other case did well and obtained a result above the threshold level. One patient blew his nose hard postoperatively and perforated his drum and developed a middle ear infection about two months after the operation. In this case the middle ear infection subsided, but a posterior inferior perforation remains. The patient, however, continued to maintain a high level above threshold three years since the complication occurred.

Two cases developed a sulfa therapy sensitivity in the course of treating their ear cavities with sulfadiazine powder. These cleared up rapidly on withdrawal of the sulfadiazine, with no more than a body rash and mild temperature elevation for a few days. One case became sensitive to penicillin. This was noted two days after the packing was removed; that is, seven days after operation. The cavity became filled with a purulent drainage and it looked as though the flap would probably slough out. This case was placed on sulfadiazine therapy and the cavity healed up rapidly and continued on as one of the best of the completely rehabilitated cases.

Tinnitus is alleviated in the majority of the cases following the making of the fenestra, but when and if it does recur it is frequently a sign of beginning closure of the fistula.

Vertigo has never been persistent in any of the cases in this series. Most cases have marked vertigo for two to three days and on the fourth to fifth day are up and walking about the ward. One case complained of vertigo on sudden change of motion for three months. This case continued on to the rehabilitation level.

The effect of infection on postoperative healing is well demonstrated by the case of a man, age 43, with a family history of otosclerosis. His audiogram indicated a mixed type of deafness with a rather marked loss of high tones. The 2048 frequency threshold was down to 35 db for bone conduction.

This patient showed no improvement at the first postoperative audiogram, done three weeks following the fenestration. He noted very little subjective improvement until two and one-half months had elapsed, at which time a badly diseased tooth root was removed. A few days following this, he noted marked improvement. The audiogram done one week after tooth extraction showed a dramatic improvement in the threshold level, which improvement continued until the end of the first year, and this gain has held its level through his third-year test.

Table 1 shows the average decibel level in the critical frequencies for both air and bone conduction throughout the course of his postoperative period. This demonstrates the fact that in certain cases a toxic focus can impede an immediate improvement.

TABLE 1.

Average decibel level for critical frequencies:	AC	BC
Preoperative, 8-1-45	65	8.3
Postoperative, 8-20-45	53.3	23.3
Post-removal of diseased tooth root, 10-22-45	33.3	—16.6
One year postoperative, 8-5-46	25	—10
Two years postoperative	21.6	— 3.3
Three years postoperative	20	5

One case came to autopsy 16 days post-fenestration. This was a man, age 50, with a bilateral progressive deafness, who had had a past history of mild hypertension and kidney dys-

function. He was checked by the internist and genitourinary specialist and pronounced fit for operation. His first five days after the fenestration were uneventful. He noted considerable subjective sense of hearing improvement. On the fifth postoperative day he developed a spiking temperature and mental changes. This was followed by cardiac and chest complications. At autopsy he was shown to have massive thrombi in the pulmonary, cerebral and leg veins with myocardial infarction, cerebral degeneration and cystitis.

This case made it possible to obtain the microphotographs of the temporal bone showing the fenestra and the flap. They also show the close adherence of the skin flap to the membranous labyrinth. The flap itself appears fairly thick. Fowler[13] maintains the thickness of monkey flaps as being associated with postoperative edema. In this case the thickness is probably in part due to the proximity to operation and also to the congestion associated with his venous thrombosis. Fig. 1 shows the flap over the ampullated end of the membranous horizontal canal. Fig. 2 shows the flap over the membranous ampulla itself.

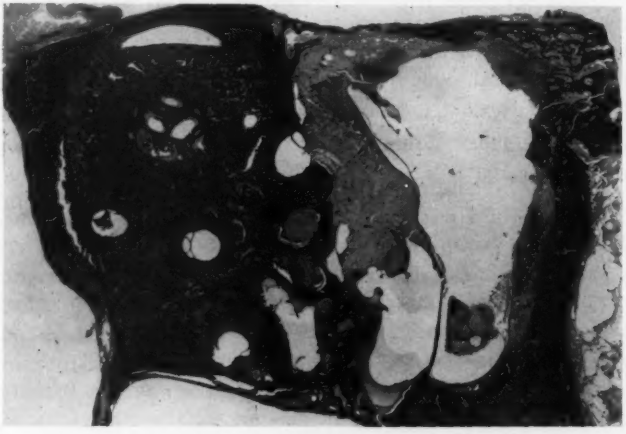

Fig. 1. Microphotograph of temporal bone showing the flap covering the fistula over the ampullated end of the membranous horizontal canal.

Fig. 2. Microphotograph of temporal bone showing the fistula over membranous ampulla itself.

HISTOLOGICAL REPORT.

(Submitted by Dr. Franz Altmann.) The histological examination of the left temporal bone in vertical serial section showed a typically located otosclerotic focus on the anterosuperior circumference of the oval window. The fistula extends from the anterior part of the ampullated end of the horizontal canal over the ampulla itself. Its margins are smooth and there are no reactive changes in any of the three layers of the capsule.

The flap is continuous with the drum and everywhere adherent to the surface of the bone except where it has been pulled away during the preparation of the specimen. The flap fills the defect of the bony capsule completely without protruding to any noticeable degree into the lumen of the perilymphatic space. The flap is covered with stratified squamous epithelium which from the margins of the flap grows over the adjacent areas of the operative cavity. The flap consists of corium, subcutaneous connective and some fat tissue. There is marked edema and vascular congestion of

the tissues with small perivascular extravasations in some places. In some areas perivascular round cell infiltration is noted. Nowhere is there evidence of new formation of bone. In one place a tiny splinter of old bone is seen on the undersurface of the part of the flap which covers the fistula.

The meso and hypotympanum show moderately marked hyperplastic changes in the mucosa with formation of folds and strands of organized tissue, particularly in the window niches. In addition there are moderate acute inflammatory changes and some blood and seropurulent exudate in the middle ear cavity.

The ampullar end of the horizontal membranous canal is adherent to the undersurface of the flap. The ampulla itself is separated from it.

The membranous endosteum at the margins of the fistula appears intact and there are no bony splinters in the perilymphatic space. The vestibule contains some blood. There is evidence of a subsiding mild diffuse serous labyrinthitis.

Sections through the cochlear show marked postmortem changes but no evidence of any pathology.

METHOD OF REPORTING END-RESULTS.

The reporting of end-results by various surgeons doing the fenestration operation has not been uniform. This has resulted in considerable confusion in evaluating comparative studies of the different operators.

The report by Lempert,[14] in 1945, and of Sullivan,[15] in the same year, suggested a division of the cases into four groups, namely: *1*. Complete rehabilitation. *2*. Partial rehabilitation. *3*. No change. *4*. Those cases which became worse. A category of four such groups was suggested by Altmann and Woodman,[16] in 1948, in which they emphasized the importance of the 2048 frequency in those cases classified as partially rehabilitated.

A recent personal communication was received in answer to an inquiry from Dr. Philip Meltzer,[17] who is a member of the committee appointed for the purpose of establishing criteria for reporting the end-results of fenestration cases. He stated that it had been suggested that a subdivision of the partially rehabilitated cases be made into two classes depending upon the decibel gains in the critical frequencies.

Our own cases were divided into four groups. This grouping follows somewhat the suggested pattern of the aforementioned operators. This survey of hearing results was compiled from 50 cases, all at least one year old and some over three years postoperation. Their average time was 22 months after operation.

The cases were grouped as follows:

Group I: All cases which had improved to 30 db or better in the critical frequencies. These cases were considered to be economically and socially completely rehabilitated.

Group II: All cases which had improved above the preoperative level but not to the full degree of Group I.

a. Where there has been more than 10 db and less than 15 db average improvement for the critical frequencies.

b. Where there has been 15 db or more improvement for the critical frequencies but not reaching the level in Group I.

Group III: Cases where the hearing is unchanged or does not show more than 10 db gain in the critical frequencies are reported as "unchanged."

Group IV: Cases where hearing became worse by 10 db or more than the preoperative level in the critical frequencies.

Results summarized: Group I, 34 per cent. Group II, *a.* 10 per cent; *b.* 16 per cent—26 per cent. Group III, 26 per cent. Group IV, 14 per cent.

Groups I and II, representing the complete and partially rehabilitated cases, totaled 60 per cent of the series.

BONE CONDUCTION IN THE OPERATED EAR.

Some gain in bone conduction was recorded from the operated ear for 85 per cent of all the rehabilitated cases (Groups I and II).

Comparison was made between the preoperative and the twelfth month postoperative audiograms.

The average gain for all cases was 7.4 db for the three critical frequencies. The greatest gain was most often registered in the 1024 frequency, followed by the 2048 frequency. Juers[18] shows a bone conduction gain in these frequencies of 6.7 db in a group recorded three months after fenestration. He also noted the 1024 and 2048 frequencies as showing the greatest decibel gain. R. R. Woods,[19] of Dublin, recently reported on bone conduction gain following the fenestration operation. In a small series of five early cases he found a 14.2 db gain in the critical frequencies. In our series, 46 per cent of the cases showed an average of 14 db gain.

THE CONTRALATERAL EFFECT.

Following the fenestration operation, some patients claim that they hear better in the unoperated ear than they did prior to their operation. This same phenomenon has been observed by Sourdille,[6] Kopetzky,[20] Holmgren,[21] Venker[22] and others.

Although we have not obtained frequent early audiograms of patients who claim they hear better in the unoperated ear, our own voice tests and the patients' reaction to sounds not heard before has been rather convincing that in some cases there is an improvement for air conduction in the unoperated ear.

The patient may insist on this improvement being present a day or two after the operation, when, and in spite of the fact that, the operated ear is packed and a complete mastoid dressing and bandage covers the operated ear. Holmgren[21] and Sourdille[6] stress this phenomenon as being present early but not being of a lasting quality.

Sourdille[6] stated: "In the case of the nonoperated ear, a certain number of patients consistently, after opening the labyrinth and for a few days after, have a notable improvement in the nonoperated ear. Does this represent an ear to ear reflex? Is this the result of a trophic or vascular reaction? This change for the better is generally of short duration and is generally followed by a progressive loss. In certain cases, however, this opening of the labyrinth seemed to curtail the progressive loss in the unoperated ear. This phenomenon is of interest only if we are able to show that the reaction of the nonoperated ear is really the result of the work done on the first ear."

Holmgren[21] noted a few cases with early improvement which he confirmed with voice and tuning fork tests and found a gain for both air and bone conduction.

The fact that this subjective sense of improvement seemed associated with the early postoperative phase only might be explained by the fact that the operated ear, while undergoing dressings and postoperative repair, with the attendant accumulation of secretions, had not reached sufficient level of improvement to take over the hearing and become as it does later, when improvement occurs, the ear with which the individual focuses his attention on sounds. Once the operated ear is the better ear, the subjective sense of any hearing improvement in the unoperated ear would be expected to disappear.

Kopetzky,[20] in 1939, commented upon the presence of a contralateral effect and stated that: "The cortical centers might be stimulated by the reactivation of the operated ear and cause an increase in sensory perception in the unoperated ear." This would imply a tonic effect on the cochlear nuclei. The fact that it is subjectively short lived would make it look like a compensatory action until the operated ear improves and takes over.

Venker,[22] in a series of 12 fenestration cases, reports audiograms showing improvement in hearing in the unoperated

ear. He seeks an explanation of this in the otosclerosis theory of Gray. He reasons that the fenestration allows greater volume of sound to penetrate into the cochlea so that the stimulus for the vasomotor system becomes more intense. The unilateral stimulus has its effect on both ears due to the fact that the sympathetic fibres of both originate in the plexus covering the circle of Willis. He states the proof of this is seen in the vascular improvement noted in the drum of the unoperated ear.

The insistence on the part of the patients of early improvement in the unoperated ear seems, in a few cases, to be accompanied later on by an actual gain in pure tone tests of bone conduction in the unoperated ear.

The cases used for the study of postoperative bone conduction were those of Groups I and II of this survey, these being the cases which were considered partially or completely rehabilitated. Averages were obtained by taking three of the postoperative periodic audiograms, including the twelfth month audiogram. All frequencies between 256 and 8190 were included.

Some improvement in bone conduction in the unoperated ear was noted in 86 per cent of the cases. Both Fowler[23] and Holmgren[24] caution against errors resulting from repeated testing, also the subjects' psychological desire to hear better and hence concentrate better. These factors may well account for many of the cases which showed only slight improvement, but there remain a few cases, approximately 15 per cent, with an average decibel gain of 13.5 in the unoperated ear. Even though this is a relatively small percentage, it would seem there are cases which show a definite gain in bone conduction in the unoperated ear which has lasted for a twelvemonth period.

Are these findings the result of repeated testing and the psychological desire on the part of the patient to hear better, or is there some tonic action along the cochlear pathways

which travel from the operated labyrinth to both the homo- and contralateral temporal lobes and then on to the contralateral labyrinthine nuclei?

The work of Walzl[25] has demonstrated that there are definite pathways between each cochlear labyrinth and its homeand contralateral temporal lobe. He shows that there is a spatial frequency localization in the cochlea and that the pattern set up in the cochlea by a sound is projected to dual auditory areas in each hemisphere. McCulloch[26] has also demonstrated the localization of the point to point interconnecting pathways from one auditory cortex to the other via the corpus callosum.

In the case of the vestibular component of the VIIIth nerve, the effect of tonic impulses from one temporal lobe affecting the opposite vestibular nuclei has been demonstrated on animals by the work of de Barenne, Dusser and de Kleyn.[27] These findings were later applied to the diagnosis of cerebral lesions in work on humans by de Kleyn and Versteegh.[28]

Further confirmation of this work in humans by the application of the caloric tests which demonstrated the value of the directional preponderance of caloric nystagmus as a diagnostic finding in temporal lobe lesions has been reported by G. Fitzgerald and C. S. Hallpike.[29]

Cawthorne, Fitzgerald and Hallpike[30] discuss the compensatory mechanism of Bechterew in which the vestibular nuclei take over and compensate for the loss of tonic impulses resulting from a utricular paresis which follows labyrinthectomy. They demonstrate that any depressing or paralytic effect on the utricle is followed by a preponderance of caloric nystagmus to the side of the normal labyrinth.

We might ask the question as to why the effect of definite improvement (13.5 db bone conduction gain) is no more frequent than in 15 per cent of the rehabilitated cases. Is the contralateral effect only an early one and are these few cases which showed lasting bone conduction gain those that just

happened to be tested at times in which the phasic reactions were favorable? It is known that in tests of preponderance reactions of the vestibular nuclei, the phasic quality shows great variability, depending on the time tested after labyrinthectomy (see Fig. 4). This phasic variability, character-

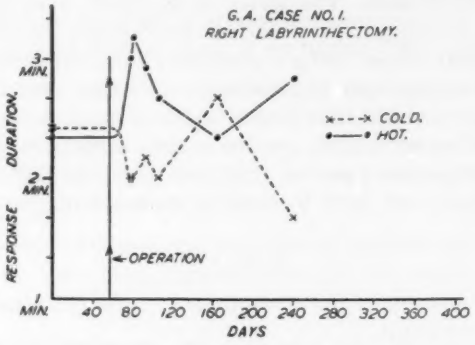

Fig. 3. Case 1. Course of caloric reactions before and after right labyrinthectomy. (From Brain, Vol. 65, 1942.)

istic of the compensatory activity of the vestibular nuclei, might account for the considerable variation in bone conduction testing postfenestration, if any analogy between the vestibular and the cochlear nuclei can be applied.

COMMENT.

My impression is that there is a definite early improvement of hearing in the unoperated ear. This effect would seem to me to be a central tonic response to the stimulation caused by the fenestration operation. This effect is compensatory and transient in character.

The few cases of bone conduction gain of lasting nature may be explained as being coincidental. If they are the result of some central tonic action, then the reason for their being so few in number may be based on the already mentioned phasic quality of postoperative testing similar to that noted in preponderance tests of caloric nystagmus resulting from

stimulation of the vestibular nuclei. It is of interest that those cases which showed a lasting bone conduction gain were not always cases which had noted an early subjective improvement.

The weight of evidence is not strong enough to merit any definite conclusion. The series is too small, even though the evidence is suggestive of such a tonic action on the part of the cochlear nuclei being a possible etiological factor in producing contralateral improvement. Further studies of this particular problem in a larger series of cases with more frequent audiometric tests, carried on in the early as well as the later postoperative period, may help either to elaborate upon or eliminate any such theoretical explanation.

SUMMARY.

1. The history of the development of the fenestration operation is reviewed.

2. A survey of the experiences during the past three and one-half years with 75 cases is presented, describing the method and modifications used, together with postoperative care.

3. The complications encountered and their sequelae are noted, together with an autopsy report with histological findings in the temporal bone.

4. One case showing the effect of infection on postoperative hearing is presented with table of the decibel average for critical frequencies before and after the removal of the infection.

5. The method of reporting the end-results of fenestration operations is discussed and the four classified groups used in this survey are described.

6. Improvement in bone conduction in the operated ear is mentioned and compared with similar findings and results of other operators.

7. The contralateral effect of improvement in the unoperated ear has been recorded by Sourdille, Kopetzky, Holmgren, Venker and others.

8. Sourdille and Holmgren have reported this effect as only early and transient.

9. Holmgren reported contralateral improvement for both air and bone conduction checked by tuning fork tests.

10. Our own experience resulted in many cases which presented this early subjective contralateral improvement, together with a small group (15 per cent) which showed a definite (13.5 db) improvement in bone conduction which lasted during the first 12 months.

11. The contralateral and homolateral pathways to the cerebral cortex have been demonstrated by the recent work of Walzl.

12. The contralateral effect in the case of the vestibular component of the VIIIth nerve has been demonstrated by the work of Cawthorne, Fitzgerald and Hallpike.

13. An attempt is made to draw an analogy between the tonic contralateral effect as seen in the case of the vestibular component and apply a similar explanation to the effect on the cochlear nuclei and their contralateral pathways which follows the fenestration operation.

14. Our impression is that the early transient contralateral effect is a central tonic effect resulting from the fenestration operation and that this is of a compensatory character.

15. The lasting bone conduction improvement noted in a few cases is regarded as coincidental or possibly the result of a phasic variability similar to the compensatory activity of the vestibular nuclei following labyrinthectomy. The latter is explained on the basis of the Becterew mechanism.

16. This series is too small for definite conclusions, but the results strongly suggest a central tonic contralateral effect. The attempt to explain this by drawing the analogy between

the vestibular and cochlear components of the VIIIth nerve may be elaborated upon when studied in a larger series with more frequent audiometric testing for both the early and late postoperative stages.

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SURGICAL REPAIR OF CONGENITAL CHOANAL ATRESIA.

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Definition: In congenital atresia of the choana narium, a persistent wall of tissue obstructs the posterior infundibulum. The atresia may be unilateral or bilateral; partial or complete. In 90 per cent of cases the obstructive wall is osseous in nature;[1] it may, however, be membranous or combine both characteristics. Otto[2] first described the condition in 1829.

Complete bilateral atresia constitutes a pediatric emergency: it is impossible for the infant to suckle and respiration is seriously impeded. Richardson[3] reported a case of asphyxia neonatorum due to this cause.

Fortunately this extreme type is rare. Even in its unilateral or incomplete form, however, choanal atresia inhibits normal development by interfering with respiration and alimentation.

Anatomy: The choana narium is bounded inferiorly by the horizontal plate of the palatine bone, medially by the posterior border of the vomer and the nasal crest of the palatine bone, superiorly by the vomerine ala and the body of the sphenoid, and laterally by the median plate of the pterygoid process of the sphenoid bone and the perpendicular plate of the palatine bone. In the lateral wall lies the pterygopalatine canal containing the palatine artery and nerves.

Etiology: The factors responsible for choanal atresia have yet to be definitely proved. According to the prevailing opinion, the abnormal tissue is the vestige of one of two

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fetal structures: the bucconasal or the buccopharyngeal membrane.[4,5,6] The anteriorly located osseous obstruction is thought to arise from the bucconasal membrane; the more posterior, membranous type from the buccopharyngeal.

A less generally accepted theory holds that the atresia results from an overgrowth of the horizontal and vertical processes of the maxilla. Boyd defended this hypothesis by pointing out the presence of islets of cartilage in the bone; these would not be present in bone formation from the mesenchyme within the membrane; however, this hypothesis fails to explain the localization of the obstruction or the fact that the membranous type is usually more posteriorly located than the osseous.

Stewart[7] believed both theories would have to be drawn upon for a complete explanation. The condition is aggravated by sinusitis or other irritants.

Symptoms and Diagnosis: In complete bilateral occlusion, severe asphyxial symptoms facilitate diagnosis; the infant is cyanotic, with cheeks and lips indrawn. Temporary relief occurs on crying, when breathing is automatically carried on through the mouth. Suckling is impossible and the child must be fed by medicine dropper until it learns mouth breathing or surgical relief is obtained.

While unilateral occlusion presents less sensational symptoms, it also interferes with respiration and nursing. During the latter the child must be held with the occluded nasal fossa in an inferior position.

The presence of a suspected obstruction can be verified by exploration of the nasopharynx and posterior nares with the finger. A probe passed into the nasal fossa ascertains the precise location of the atresic wall and the degree of impermeability. By blowing air through the nostrils with a rubber bulb, the patency of the nasal passages is determined.

On examination with the nasopharyngoscope or laryngeal mirror, the bony type of obstruction appears white, while the membranous (or the membranous center in the mem-

brano-osseous type) appears translucent and dappled. The obstructed nasal fossa is usually filled with a sticky mucus substance which can be expressed from the nostril.

The obstruction can be demonstrated Roentgenologically by the instillation of lipiodol into the nasal fossae.[8] In cases of unilateral occlusion there is usually a marked septal deflection toward the obstructed side.[9] The septum tends to comparative straightness in bilateral atresia.

Differential Diagnosis: Choanal atresia is readily differentiated from other intranasal obstructions. While large adenoids may cause partial stenosis in the infant, they do not

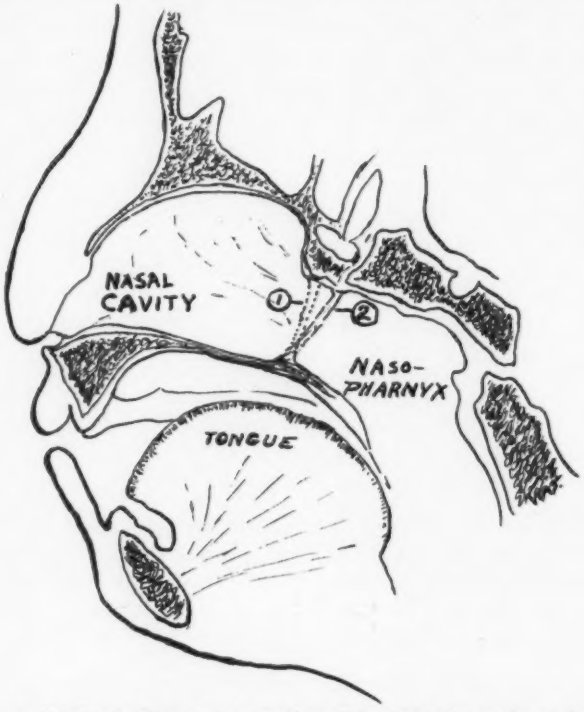

Fig. 1. Schematic diagram of a section of the head of a newborn showing (1) location of a typical osseous occlusion, and (2) location of a typical membranous occlusion of the nasal choana.

produce complete occlusion. It is interesting to note that adenoids are usually absent in bilateral atresia, but Geiger and Roth[10] reported their presence in a case of unilateral occlusion.

Pyhsical examination suffices to distinguish choanal atresia from such other obstructions of the posterior nares as congenital postnasal polysp and encephalomenigoceles. This is true also of blood clots and other products of birth which

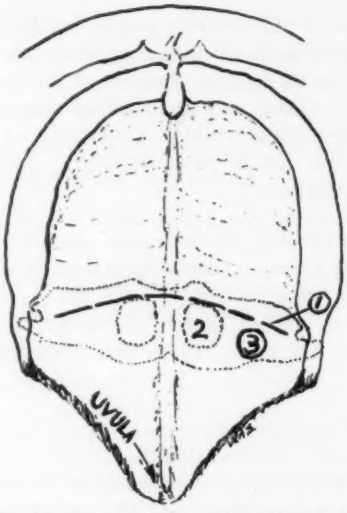

Fig. 2. The oral palate. (1) Line of incision in the palatine mucosa. (1) Fenestration of the os palatinum. (3) Os palatinum outlined in dotted lines.

may have been pushed or drawn into the nostrils during delivery. Congenital atresia of the nasopharynx may simulate postnasal imperforation, but here again examination will dispel any doubt.

Surgical Repair: The surgical repair of choanal atresia dates back to 1853, when Emmert[11] operated successfully on a seven-year-old boy. Since then many methods have been employed to eliminate the obstruction.

If an emergency the atresic wall, whether osseous or membranous, can be perforated to permit nasal breathing. No matter how large the opening made, however, it has a tendency to close on cicatrization. Morgenstern resorted to repeated excisions to maintain the fenestra.

The obstruction can be attacked anteriorly by the intranasal route[12-17] or posteriorly by the nasopharyngeal. Morgenstern approached it from both directions, using surgical diathermy.

When the size of the anterior nares and nasal fossae of the infant is considered, it is apparent that the intranasal approach leaves much to be desired. The surgical field is uncomfortably confined and the desired correction cannot be carried out under direct vision. There will of necessity be a large area of scar tissue, with resulting adverse effects on physiological function.

While the Kazanjian trans-septal operation largely overcomes these objections, extensive septal surgery is inadvisable in the infant in view of its possible effects on the developmental processes of the nose. Similarly, one hesitates to invade the infant antrum, as in the transantral method of Wright, *et al.*[18]

The more expeditous transpalatine route was used by Blair,[19] Steinzeng,[20] Schweckendiak,[21] Neto[22] and Ruddy.[23] Their techniques vary in the position of the incision and the amount of hard palate removed. Blair's incision — from the anterior part of the palatal mucosa to the junction of the hard and soft palates — seems unnecessarily long. Steinzeng raises an exceedingly large flap, embracing almost two-thirds of the palatal mucosa. Schweckendiak and Neto make an I-shaped incision in the posterior third of the hard palate and raise two lateral flaps. Ruddy's incision follows a horizontal flat arc, starting just medially to the orifice of the posterior palatine canal on one side and extending to the same point on the other. He raises a posterior flap with very good exposure. Both Neto and Ruddy penetrate the hard palate in two places, on either side of the septum (vomer). The operation is then

performed through the nasal submucosa. This approach permits a double attack upon the obstruction as the intranasal route may be used as a supplement.

Following excision of the atresic wall, the edges of the nasal mucosa can be dropped in place as in Ruddy's technique; or flaps of mucosa can be transposed as in one of Blair's methods; or a split skin graft can be transplanted to the raw area.[24] The transpalatine procedure of choice would combine Ruddy's incision, Neto's fenestration and Blair's treatment of the nasal mucosa, as follows:

Step 1: Palpate the orifices of the two posterior palatine canals. (These feel like two soft spots in the hard palate, just medial to the upper third molars.)

Step 2: Incise the palatine mucosa and periosteum in an anterior arc between these two points.

Step 3: Elevate a posterior flap subperiosteally for about 1 to 2 cm.

Step 4: Locate the atresic wall and remove sufficient palatine bone from either side of the midline to facilitate the attack on the obstruction.

Step 5: Remove the entire obstruction, through the nasal submucosa insofar as possible. (A small blunt instrument, inserted into the nasal fossa, expedites this step.)

Step 6: Excise redundant mucous membrane or transfer mucosal flaps to defect.

Step 7: Insert rubber obturator of as large diameter as possible into nasal fossa to hold the tissues in place.

Step 8: Cut a piece of gelfoam to fit the palatal defect. Insert into defect.

Step 9: Close the incision in the palatine mucosa with interrupted catgut sutures.

Several types of obturators have been suggested, such as stent, tin, rubber,[25] medicated gauze strips[26] and acrylic.[27] One of us (Sheehan) prefers stent as it is always available, read-

ily molded, nonirritant and not easily affected by body heat. It should be mixed one-third white and two-thirds brown as the white gives added stability to the mold.

Rubber also is efficient and easily obtainable; old catheters can be used. When the rubber-tube obturator is employed, it should be slid back and forth gently each day.

Obturators are dispensed with after two weeks. It cannot be overemphasized that adroit after-care is the most important single feature of treatment.

CONCLUSIONS.

1. It is fairly well established, though not definitely proved, that congenital choanal atresia arises from the bucconasal or buccopharyngeal membrane.

2. While slightly more difficult in some respects, the transpalatine route affords the most satisfactory surgical approach.

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DETECTION OF NOISE SUSCEPTIBLE EARS.*

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INTRODUCTION.

In the literature, frequent reference is made to the fact that different individuals exposed to the same amount of noise display different degrees of resultant hearing loss. Such findings clearly imply differences among individuals in susceptibility to noise-induced trauma. Stated simply, if two individuals, A and B, are exposed to the same noise for the same length of time, A may develop a significant and permanent hearing loss, whereas B may not.

The locus of loss, when it occurs, is no longer in doubt. Many studies exist to show that noise trauma is related to alterations in cochlear structures. Although the bulk of the evidence is drawn from animal studies, there is no reason to doubt its application to the human ear. In one of the most recent publications on this subject, Ruedi and Furrer[6] have shown histological evidence, in fresh postmortem sections, of severe damage to human ears following exposure to blast. Hemorrhage within the internal ear and bleeding from the canal, together with demonstrable disruption of cochlear elements, are probably evidence of irreversible impairment in the portion of the auditory range damaged by explosion, blast,

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concussion, etc. Survivors of such episodes usually display a hearing loss as determined by functional tests, but these individuals sometimes show a recovery of some fraction of the loss when their audiograms are followed over a sufficient length of time. Individuals who work in noisy environments, such as factories, develop hearing losses which, on the average, are proportional to the intensity level of the noise and the length of time of exposure to the noise. Individuals having normal hearing who are temporarily exposed to high sound levels develop a loss which disappears not long after termination of the exposure.

All of these considerations indicate that noise exposure probably initiates a continuum of events, ranging from a reversible temporary loss for short exposures at moderate levels to a permanent irreversible loss induced by exposure to excessively intense sound pressures, such as those produced by blast, explosion or concussion. The most challenging aspect of the above summary is the determination of the point at which the loss ceases to be reversible and becomes a permanent impairment. It is precisely this determination which is involved in the matter of susceptibility, since prediction of susceptibility obviously implies prior knowledge about a nonexistent loss which will become manifest as the result of noise exposure.

THE MECHANISM OF IMPAIRMENT.

It has been brought out that actual histological damage can be demonstrated under extreme exposure conditions and the hypothesis is made that such damage is irreversible. Functional tests, however, demonstrate the reality of reversible losses. The question may now be raised as to the physiological basis for a reversible loss. An excellent paper by Hamberger and Hyden[1] gives one such mechanism in explanation of this phenomenon. These experimenters exposed a series of animals to sound pressures graded in level and duration of exposure time; in postmortem sections, they studied alterations in the nuclei of cochlear ganglion cells. These altera-

tions were demonstrated by delicate biochemical and biophysical techniques, utilizing absorption spectra, ash residue and dye indicators. Their results may be summarized as follows: intensity and exposure time are directly related to corresponding depletion of specific protein compounds in the cell nucleus and nucleolus. Norms were established for the concentration of these substances in unexposed ears. It was shown that animals surviving 21 days following the exposure period had cells of normal concentration; those sacrificed in intervening periods had concentrations varying with elapsed time. In a later series of experiments, Hamberger and Hyden demonstrated that the conditions which produced these metabolic changes also produce functional hearing losses. A strong relationship between this particular aspect of cell metabolism and functional response is certainly suggested. The logical deduction from this research is that functional losses become irreversible at some point in the process where the resting cell is no longer capable of restoring the normal concentration of protein compounds.

It is, of course, not possible to state with certainty that the phenomena described above completely explain the mechanism of reversible or irreversible impairment in the human ear. The temporary threshold shift demonstrated in humans following noise exposure usually returns to normal threshold after a few hours and, at most within three to four days, whereas the metabolic changes studied by Hamberger and Hyden in the guinea pig were detectable up to almost three weeks following exposure, with maximum depletion at 14 days.

INDICES OF SUSCEPTIBILITY.

It is probable that every ear when exposed to a sound of sufficient intensity over a period sufficiently long in duration will undergo some functional loss in acuity. The degree of loss varies widely in individuals, as previously stated. There appears to be, therefore, a continuum which, for a sound of given intensity level and length of exposure, includes all indi-

viduals ranging from those who show only slight losses in acuity and rapid recovery to those who show large losses and long periods necessary for recovery.

In this paper, attention is focused upon the latter end of the continuum. The noise susceptible ear, in this context, is obviously one which displays significantly large losses of acuity in response to noise exposure and requires a long time for recovery to normal. When the *recovery time* required approaches infinity, this ear has undergone a permanent and irreversible decrement in acuity. In the industrial world, an individual having such an ear should not be exposed[2] to high noise levels for long periods; if he is so exposed, he will unquestionably suffer permanent loss of acuity in some or all portions of the auditory range. That such noise susceptible individuals exist is a fact amply confirmed by repeated observation and by reports in the literature; that such an individual can be identified prior to the actual establishment of the loss is, however, an hypothesis yet to be confirmed.

Peyser,[3] writing on this subject in 1940, advanced the idea of test bombardment with subsequent threshold shift as an index of susceptibility. His original test consisted of two thresholds at 256 cycles, one taken prior to exposure at that frequency for five minutes, the second threshold measured 30 seconds after cessation of the exposure tone. Peyser claimed that individuals whose second threshold was 5 db or more higher after exposure were noise susceptible. No validation of this claim was shown, in his paper, by tests of these "susceptible" ears under actual noise conditions. Wilson[5] applied Peyser's principle in studying soldiers before and after exposure on rifle and pistol ranges. He used an 80 db exposure tone of 256 cycles with a maximum duration of eight minutes, delivered by a Western Electric 6A Audiometer; the entire audiogram (nine frequencies) was measured both before and after exposure to the 256 cycle tone. Wilson considered shifts of 10 db or more at any frequency as a criterion of susceptibility. Out of 85 subjects, 27 showed susceptibility (accord-

ing to this criterion) by the audiometer test. All 85 subjects were next sent to the firing range; of the 27 predicted to be susceptible by advance testing, 21 showed criterion loss or greater after retesting following exposure to rifle and pistol noise. Ten other subjects not predicted susceptible also showed criterion loss or greater. In a later series of experiments, Wilson[9] used 2048 cycles at 80 db for eight minutes as the exposure tone and studied a group of 108 soldiers; his results, so far as prediction is concerned, were approximately the same as those of his original study. Wilson concluded that practical predictive test can be constructed by careful selection of an appropriate exposure tone.

Peyser[4] has revised his original procedure and now advocates an exposure tone of 1000 cycles at a level of 100 db for three minutes. The 100 cycle threshold is measured first, then the ear is bombarded for three minutes; 15 seconds after cessation of the exposure tone, the 1000 cycle threshold is redetermined. Peyser considers that ears which show shifts of none to 5 db as a result of this test are resistant to noise, ears which show 5 to 10 db shifts are borderline, and ears which show 10 db or greater shifts are "hypersensitive" or susceptible to noise impairment. In Peyser's paper, no data showing the subsequent effects of industrial noise on these ears are given.

Investigators writing on the subject of noise impairment[2,4,8] usually regard an already existing hearing loss as contraindicative to the advisability of noise exposure. Although there is some question about this point when it involves middle ear type losses, there seems to be general agreement regarding individuals with inner ear pathology. Larsen[2] says:

"Last, but not least, an important prophylactic measure is the selection of laborers who shall work in a noisy shop. Persons who suffer from a disturbance of perception ought not to be employed in a noisy works, for it must be anticipated that a debilitated sense organ responds far more rapidly, more intensely, more extensively, and under easier fixa-

tion of the injury, than does the sound (normal) ear. The same applies to laborers presenting predisposition to ear disease. As was mentioned before, they will likewise react more readily to the influence of noise than normal persons."

PREDICTIVE TESTS.

The methods described by Wilson and Peyser have been given added importance by Hamberger and Hyden, in that a physiological basis has been supplied by the latter authors; but the future development of an accurate predictive test depends upon careful standardization of technique.

It appears that the test bombardment of ears, followed by measurement of the resulting threshold shift, will form the basic technique in a predictive test. The first question to be resolved in the establishment of a standardized test is the selection of the stimulating or bombarding sound. Nearly all of the many studies reported in the literature have been conducted with pure tone stimuli; unless repeated audiograms are to be taken, one must obviously choose a single frequency as the stimulus in a predictive test. Although the effects of exposure to a loud pure tone are not always confined to that frequency and/or immedately adjacent frequencies, there is always the possibility that any single pure tone may be too specific for use in a generalized predictive test. A more obvious approach would be to bombard the entire auditory range simultaneously. For this purpose, a suitable type of stimulus is available. Thermal noise, or "white" noise, is an easily produced stimulus whose chief characteristic is statistical equality of energy at all frequencies in the auditory range. Until more information is available about the results of predictive testing, the writer believes it more practical to employ a complex stimulus which allows no portion of the auditory range to "escape" exposure. It is, perhaps, worth observing that industrial noises are always complex; in a test, thermal noise would have higher face validity than pure tones.

The next question involves the frequencies to be studied in the postexposure audiogram. The evidence from many reports in the literature leaves little doubt that the more vulnerable portion of the auditory range lies in the high frequency end; *i.e.*, above 1000 cycles. Time is needlessly wasted, therefore, in measuring postexposure thresholds at low frequencies in a practical test of susceptibility. For the time being, at least, it would be more profitable to concentrate attention on the 2000 to 8000 cycle range, with special consideration being given to 4000 and 6000 cycles. It is in this region that large threshold shifts appear after short exposures.

A third question is that of the criteria used to differentiate susceptible from nonsusceptible individuals. It is suggested here that two measurements be considered:

1. The magnitude of the threshold shift; *i.e.*, the number of decibels by which the postexposure threshold is elevated above the pre-exposure threshold.

2. The recovery time; *i.e.*, the number of minutes required to elapse between termination of exposure and return to pre-exposure threshold. Of the two measures, it is conceivable that the latter will prove more highly differentiating than the former. It was brought out in the preceding discussion that, when recovery time becomes infinite, permanent reduction in acuity has been established. The temporary threshold shift is a transient phenomenon whose very title shows its dependency upon time. It would be a comparatively simple matter to establish the normal rate of recovery of 4000 cycles, for example, to pre-exposure threshold; given the norm, one could consider individuals who display markedly retarded recovery rates as potentially susceptible.

The fourth issue is that of test ventilation. It will be useful here, perhaps, to recall the reason for predictive tests. These are to be employed in segregating the *individual* who is noise susceptible. In the industrial application of such tests, the administrator must have high confidence in the meaning of his results, for they might influence strongly the placement of

the applicant for any job involving high noise levels. It is, therefore, necessary that any predictive test be shown to be valid by actual results in the field before such tests can be applied generally. The method of establishing validity is essentially that employed by Wilson, discussed in the preceding section of this paper. The test must be given in advance to a group of individuals about to take employment in a noisy environment. The hearing acuity of this group must be followed over the period of time necessary to establish significant, and presumably permanent, losses in certain individuals. If, then, these individuals are those predicted to be susceptible by the original test, and, *if no individual not predicted susceptible* develops losses of the same order, the validity of the test will have been supported.

SOME PRACTICAL PROBLEMS.

When the basis for predictive tests has been confirmed, as suggested above, there will remain the necessity for a standardized presentation. The critical application of such tests requires that as little room as possible be left for erroneous interpretation.

It will probably be necessary to take into account the tinnitus which usually follows when ears have been exposed to noise. Peyser comments upon this disturbance but says that his 1000 cycle exposure tone seems to produce tinnitus of a short duration, about 15 seconds. In a study of the temporary threshold shift, the writer exposed 12 normal hearing subjects to thermal noise at 105 db above reference level (.0002 dynes/cm.^{2}) ; these subjects reported the presence or absence of tinnitus following exposure and attempted to match its pitch on a variable frequency oscillator. Table 1 shows the frequency chosen as representative of the pitch of tinnitus by these subjects; since tinnitus is probably not a pure tone in most subjects, the frequency here should be regarded as specifying the region in which tinnitus typically occurs, but not its exact frequency level:

TABLE 1.

Subject	Frequency—C.p.s.	Comments
1	500	Slight
2	900	Definite
3	——	None reported
4	5000	Fairly definite
5	——	"Buzzing"
6	3750	Indefinite
7	4500	Fairly definite
8	1000	Fairly definite
9	5200	Fairly definite
10	6000	Definite
11	7000	Fairly definite
12	10,000	"About"

The majority of these subjects experienced some tinnitus and, for the most part, indicated it to lie in the high frequency region of the audiogram; however, no subject reported tinnitus severe enough to interfere seriously with his judgment of threshold. Since these subjects were not highly trained observers, it appears that tinnitus does not necessarily represent an insuperable obstacle to postexposure threshold measurement. It is important, however, to note from Table 1 that tinnitus seems to occur most often in the frequency region proposed for predictive tests. On this account, some provision ought to be made against its possible interference in a general predictive test. The now familiar pulse tone technique[5] seems to be well suited. As is known, this technique requires the subject to indicate how many tone pulses were heard at any given level. Consequently, the examiner is able to check the threshold by means other than the subject's unsupported statement that he heard or did not hear the tone.

In a previous paper,[7] the writer has discussed the need for careful control of time in predictive tests, because the temporary threshold shift is a transient phenomenon. Fig. 1

shows a circuit designed by the writer which will make possible a standardized presentation in which time is not only controlled but made the criterion of susceptibility.

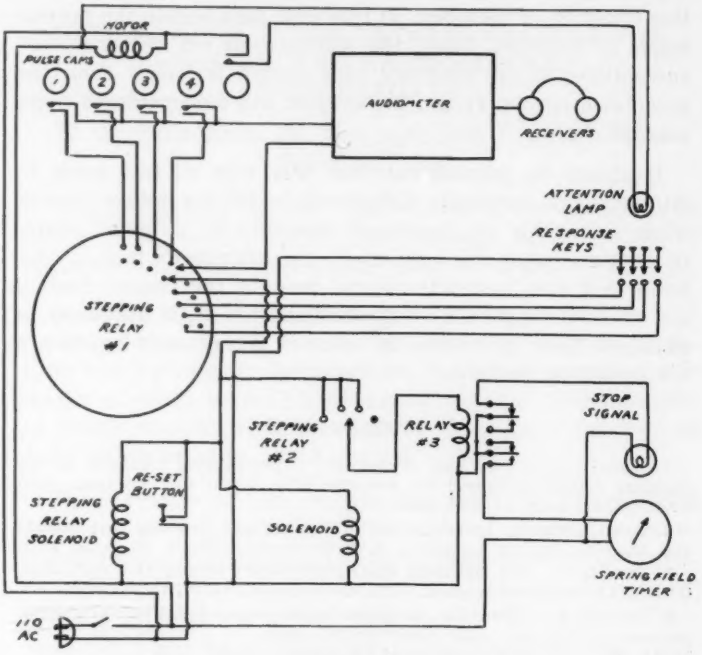

Fig. 1.

This circuit applies the pulse tone technique recommended above. The test is intended to begin immediately following a standard exposure interval to thermal noise. With the audiometer set at one of the recommended frequencies in the 2000 to 8000 cycle range, random patterns of pulse tones are automatically presented at the pre-exposure threshold level. By pushing buttons numbered according to the pulse patterns, the subject registers which number he heard. When the correct button is pushed, the stepping relay advances to a new

pattern. When the subject has correctly heard, and signaled, three consecutive patterns, the time is automatically stopped and the test ended. The timer measures the time elapsed from beginning to end of test, *i.e.*, "recovery time," according to this criterion of recovery. If this time falls within the normal range of recovery times, the subject will not be considered susceptible; if the recovery time shown is longer than the acceptable normal range, the subject will be considered noise susceptible.

It should be pointed out that this type of test tends to minimize the inevitable differences in interpretation arising when thresholds are measured manually by different examiners, since, with this equipment, the examiner's task is confined to giving instructions and reading the timer. Such a test could be applied by untrained examiners, if necessary, a situation likely to be true in industry where audiologists are not generally available.

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HEMANGIOMA OF THE LATERAL BAND OF THE PHARYNX AND NASOPHARYNX. CASE REPORT.*

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Notwithstanding the fact that tumors of the nasopharynx are common, certain rare types challenge one's method of treatment and diagnostic ability, prior to biopsy.

In determining the diagnosis and methods of treatment of masses of this type, it is important that the following definitions are considered: hemangioma is a tumor composed primarily of blood vessels; lymphangioma has a preponderance of lymph vessels; while an angioma has cells which tend to form either blood or lymph vessels.

These tumors of the nasopharynx must be differentiated from the more common growths such as sarcoma, fibroma, endothelioma, carcinoma, chordoma, craniopharyngeal cysts, as well as hypertrophy of the lymphoid tissue. Woodward[5] states that pseudoangiomas, that is, bleeding polypi, fibroangiomas and cirsoid aneurysms must be differentiated from true angiomas.

To my knowledge only two hemangiomas arising from, and limited to, the lateral pharyngeal band have been reported, one by Lillie[2] and another by Lichtwitz.[1]

Woodward,[5] Lillie,[2] Rosenberger,[4] Milligan[3] and others have also recognized the rarity of these tumors.

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With the exception of the possibility of trauma, the etiology, as in most other new growths, is unknown. It might be pertinent to think of the basic etiologic factor as an embryological mal-development.

Treatment, in general, is surgery, radiation, a combination of the two, or injection of a sclerosing agent. The growths should be removed, and when feasible, during their incipiency. One must be prepared to cope with extensive hemorrhages during any of the above procedures.

CASE REPORT.

A well developed female, age 19, when first seen was complaining of having had a full feeling in her throat for the past year. As she had had no other symptoms, she procrastinated having an examination until she saw the growth in her throat.

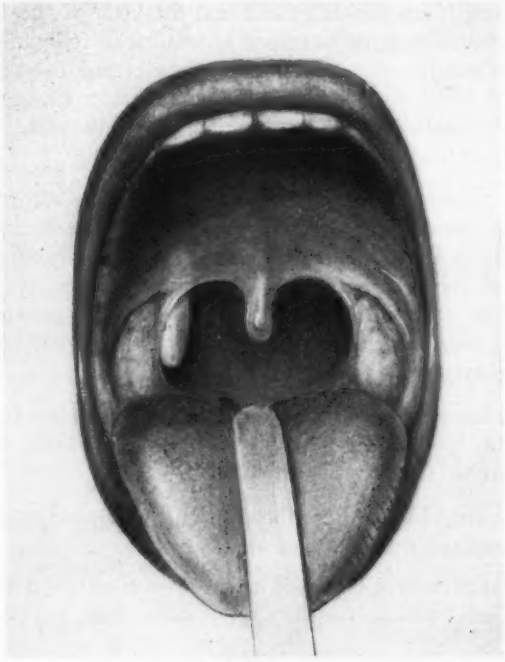

Fig. 1. The primary tumor.

An examination revealed the nares and ears to be essentially negative. The submaxillary glands were moderately enlarged bilaterally. Moderate-sized infected tonsils were present.

A mass, mulberry in color, was seen involving the right lateral pharyngeal band extending from the upper third of the tonsil to a centimeter below the Eustachian orifice. It measured approximately 6 mm. in width and 5 mm. in height. Through the tightly stretched mucous membrane covering the growth several blood vessels and capillaries were seen. These had the appearance of venous channels. The nasopharynx, including the Eustachian orifices and posterior nares, was negative. A Roentgen examination of the sinuses revealed no clouding. The cervical sympathetic was evidently not involved as there was no evidence of Horner's syndrome. A general physical examination, including serology, was negative. A diagnosis of hemangioma was made.

In determining the type of anesthesia to use for the operation, we decided to avoid infiltrating around the mass, as inadvertently the needle might penetrate the growth and cause hemorrhage; consequently, the surgery was performed by using only topical cocaine anesthetic, which proved satisfactory.

With instruments for ligating the external jugular at hand, if needed, the soft palate was retracted, thereby rendering visible the entire tumor.

Fig. 2. Secondary growth, three months after coagulation of the primary mass.

The whole mass was electrocoagulated, as well as 2 or 3 mm. of what appeared to be normal tissue immediately surrounding the growth. No bleeding occurred during the surgical procedure or during convalescence.

In three weeks the patient was dismissed as cured, having made an uneventful recovery.

Fig. 3. Photomicrograph of the secondary growth (low power).

Three months later the patient returned with a pedunculated tumor which had three lobules approximately in the same location as the first tumor. It was creamy white in color and friable upon palpation.

The entire mass was irregularly rounded and about 1.5 cm. in diameter. It was attached above the level of the soft palate, and hung downward into the oropharynx.

Under general anesthesia we attempted to dissect the pedicle from its attachment, but as each lobule was grasped for traction, it became detached from the pedicle. Finally only the pedicle remained. This we dissected from its base on the lateral pharyngeal fold. We then touched the raw area with trichloracetic acid. Little bleeding ensued. A tonsillectomy was also performed at this time.

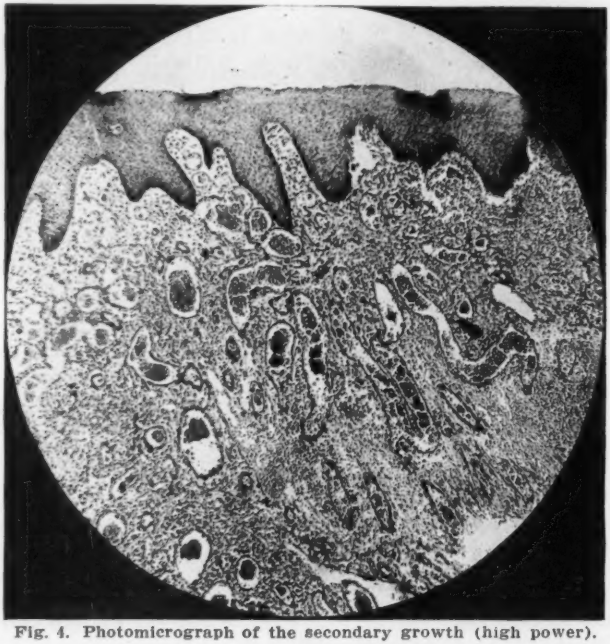

Fig. 4. Photomicrograph of the secondary growth (high power).

The report of the pathologist on the tissue removed at the second operation is as follows: "The tissues show a covering of stratified squamous epithelium of varying thickness, ulcerated in some areas and diffusely infiltrated with polymorphonuclear leucocytes. Dermal papillae and deeper connective tissues for a depth of 3 to 4 mm. are filled with vascular channels of varying size. Those channels are dilated, lined by endothelium and filled with red cells. The stroma of this vascular lesion is the site of many neutrophiles and the inflammatory reaction is most

intense at the periphery of the section. Round cells and histiocytes are present but are not nearly so numerous as are polymorphonuclear cells.

"*Diagnosis:* Hemangioma of pharynx with acute and chronic inflammatory changes presumably secondary to ulceration of the pharyngeal mucous membrane."

CONCLUSIONS.

1. The angioma type of tumors rarely occur on the lateral pharyngeal fold.

2. A surgeon should be prepared to cope with an extensive hemorrhage when removing these as well as other new growths of the pharynx.

3. These masses at times retrogress after only a portion of the tumor is removed; however, in this case all of the visible tumor was removed, yet it recurred in a totally different form.

4. After two years the patient has had no further reocurrence.

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Bennie Dillon Building.

SOLITARY NEUROFIBROMA OF THE LARYNX (REPORT OF TWO CASES).

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Although solitary benign tumors arising from peripheral nerves and possessing the characteristic picture of neurofibromata are seen frequently in almost all parts of the body, their occurrence in the larynx is extremely rare. Indeed, it is so rare that our review of the world's literature on the subject reveals only 19 such cases, nine being reported by foreign investigators and 10 reported in the American literature. The first case reported was by the German, Suchanek[1], in 1925. Jackson and Coates[3] reported the first case in the American literature in 1929. Smith[13] reported three cases in 1944, which is the largest number of cases presented by one author. New and Erich[8] found only one case in their review of 722 benign tumors of the larynx seen at the Mayo Clinic in a 30-year period ending in 1938. At the Medical College of Alabama we have seen two cases of solitary neurofibroma of the larynx since 1943.

CLINICAL CONSIDERATION.

The symptom complex presented by patients with solitary neurofibroma of the larynx is that generally found in all noncancerous tumors of this organ. Depending upon the size, position and location of the growth, symptoms vary from mild hoarseness to distressing dyspnea.

The majority of the patients reported were in the third decade of life; however, one patient reported was 60 years of age (Case 1) and the youngest was four.[15] The disease is more frequent in females than in males.

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CASES REPORTED THUS FAR IN LITERATURE.

Author	No. of Cases	Date	American or Foreign
Suchanek[1]	1	1925	Foreign
Holmgren and Bergstrand[2]	1	1928	Foreign
Jackson and Coates[3]	1	1929	American
Claus[4]	1	1930	Foreign
Vail[5]	1	1933	American
Gignoux[6]	1	1936	Foreign
Malan[7]*	2	1937	Foreign
New and Erich[8]	1	1938	American
Hoover[9]	1	1940	American
Seidel[10]	1	1940	Foreign
Baldenweck and Leroux[11]	1	1942	Foreign
Van Loon and Diamond[12]†	2	1942	American
Thomas Smith[13]	3	1944	American
New and Devine[14]	1	1947	American
Oliver, Diab and Abu-Jaudeb[15]	1	1948	Foreign

*One of the cases reported by Malan was previously diagnosed and reported by Morshik.

†One of these cases was reported by Tucker in a personal communication to the authors.

The tumor may appear in any portion of the larynx but has been found most commonly in the region of the aryepiglottic fold. Its usual appearance is that of a smooth, rounded, solid, yellowish encapsulated growth. If the tumor is small it may be removed by large cupped biting forceps, as was experienced in our two cases. As the growth enlarges and is discovered at a later stage in its development it may be necessary to remove it by laryngofissure. The largest tumor of this type, reported by Vail,[5] was $3\frac{1}{4} \times 2\frac{1}{2} \times 1\frac{1}{2}$ cm. The exact diagnosis, of course, is established by microscopic examination of the tissue removed.

PATHOLOGIC ANATOMY.

Histologically, these tumors are characteristically neurofibromata. The great confusion in terminology, such as "neurinoma, "Schwannoma," "peripheral neurofibroma," "peripheral fibroblastoma," "perineural fibroblastoma," etc., has arisen because of the highly controversial theories as to the

specific tissue from which tumors of this type have their origin and the tendency of the different pathologists to name the tumor observed after the supposed tissue of origin.

Smith[13] states, "These characteristics are best synopsized by Ewing's concise description. He stated that the tumor is covered by a distinct capsule which he thought was distended epineurium. He described the growth as being made up of two kinds of tissue, the main tissue being fasciculated, with elongated cells and fibre bands arranged in a palisade manner, and the remaining tissue being reticulated with few fibres, irregularly scattered cells and much intercellular fluid lying in vacuoles or forming small cysts containing mucinous material. The blood vessels in this tissue show characteristic hyaline thickening." Smith[13] further states, "These single noncancerous tumors, designated by so many different terms, are usually quite firm and well encapsulated, and have no tendency to undergo cancerous degeneration. They tend only to destroy by direct pressure, and as their growth is usually slow and characterized by frequent arrests, their effect on the larynx is principally that of obstruction. If the growth is entirely removed, there is no tendency toward recurrence."

CASE REPORT.

Case 1: J. W. L., bookkeeper, age 75, white male, was apparently in good health until January, 1928, when he noticed the insidious onset of progressive dyspnea, slight nonproductive cough, and a feeling of a lump in his throat that he localized by pointing to the level of the cricoid cartilage. These symptoms became progressively worse until 1938 when, in addition, he became hoarse. There was no dysphagia, hemoptysis, etc. He was so dyspneic he could hardly walk 20 feet without resting. He consulted a physician, and in the course of the next five years had three direct laryngoscopies with removal of some tissue on each occasion.

Upon our first examination, April 28, 1943, the patient appeared extremely dyspneic. Routine ear, nose and throat examination was negative except for the larynx. The patient was hoarse and external examination of the larynx revealed it to be freely movable and there were no remarkable nodes in the neck. Mirror examination of the larynx revealed the epiglottis to be normal. The aryepiglottic folds were slightly injected and the false cords edematous. There was some limitation of motion of the left cord and on the upper surface of the base of the left cord there was a pedunculated, small, yellow nodular mass about the size of a small pea impinging upon the glottic chink. The larynx was normal otherwise.

On Sept. 8, 1943, a direct laryngoscopy was done, and the mass was completely removed with cup-type laryngeal forceps, including a small part of the true cord. Since operation the patient has done well. His last visit was Jan. 6, 1949, and examination revealed no recurrence.

PATHOLOGICAL REPORT.

Microscopic Description: Section through specimen shows the bulk of the small mulberry-like masses to be made up of dense, old, hyalinized connected tissue connected by stratified cuboidal epithelium. In some areas this epithelium appears to have cilia. After examination of Zenker's sections the presence of vacuolated macrophages, plus the fact that there is a distinct tendency of the nuclei to palisade, inclines one to believe that this represents neurofibroma of the vocal cord.

CASE REPORT.

Case 2: H. H., filling station operator, age 23, white male, was first seen by us July 1, 1948, complaining of hoarseness and dry cough of one month's duration. There was no dyspnea, dysphagia, pain, hemoptysis, etc. Routine ear, nose and throat examination was negative except for the larynx, which showed injection of both cords, worse on the left, and a small, yellowish growth on the left aryepiglottic fold adjacent to the left arytenoid.

On the same afternoon the patient was laryngoscoped under local anesthesia and the mass from the left aryepitglottic fold removed *in toto* with laryngeal cup forceps. The postoperative course was uneventful and when last seen on Jan. 6, 1949, the patient was doing well without recurrence.

PATHOLOGICAL REPORT.

Gross Description: The specimen consists of a firm, gravish white mass, irregular in shape, measuring 7 mm. long and 3 mm. in diameter.

Microscopic Examination: The section through the specimen shows a surface of stratified squamous epithelium beneath which there is a peculiar plexiform structure made of bundles of fusiform cellular elements with elongated, wavy nuclei and very delicate pink-staining fibrils. In general these are parallel in their arrangement, but in some places there seems to be a tangential direction to some of the fibrils; in other areas they are in cross-direction and with phosphotungstic acid-hematoxylin stain the structure is brought out even more prominently than previously noted. Many of the fibrils are blue and are apparently of nervous origin, but many were also brownish and raised the question as to whether these cells are arising from the sheath of Swann. There is no variation in nuclear size; mitoses are practically absent.

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LIGHTNING AS A CAUSE OF HEARING LOSS.

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A review of the available literature on lightning trauma reveals a number of case reports, but few of these deal with hearing losses uncomplicated by other trauma. A thorough investigation as to the nature of lightning has been published by Harrison,[1] and for information on this subject the reader is referred to this work.

REVIEW OF LITERATURE.

Many unique and bizarre injuries have resulted in individuals struck by lightning. Hegner[2] reported compound fractures of the feet from lightning, while multiple skull fractures have been reported by Knaggs,[3] Crawford[4] and others. Lea[5] reported injury to the muscles of the eye, but by far the most common lesions of lightning discharge are burns of varying severity. Silverman[6] reported a case of unilateral deafness caused by lightning with bleeding from the ear for three days after the accident. On inspection of the drum after the bleeding ceased, no evidence of trauma to the canal or drum was seen; however, the drum was reddened. Crawford and Hoopes[4] reported a case of skull fracture with burns and injury to the ears. The right ear continued to drain spinal fluid for seven days. Thirty-four days after the injury they reported the hearing in the left ear as fair, and in the right ear as poor (almost complete nerve deafness), with marked scarring of the right drum in the posterior quadrant. Other case reports, such as that of Cook and Boulting,[7] are not too

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informative. They report a patient injured by lightning on June 14, and on July 8 the patient complained of ringing in the ears and free discharge of pus from both. It was felt that the otitis media may have been set up by rupture of the drum from the thunderclap or by prolonged exposure while in a state of collapse following the injury. One of the most detailed reports of ear injury from lightning is that of Thomas.[8] He describes a patient with superficial burns of the skin and slight hemorrhage from both ears. There was no perforation in either eardrum. "The umbo and its immediate surroundings were intensely congested and ecchymotic when first examined six days after the accident. It is very likely that there were minute holes in the tympanic membranes like the ones that are made by the spark of an induction coil upon a thin sheet of paper when held between the sparking points." Since the umbo is the most prominent area of the drum, he surmised that this suffered the majority of the injury and hence demonstrated the major pathology.

The case of otorrhea most probably represents the results of concussion with subsequent infection; however, the nerve deafness reported may be attributed to actual nerve damage. Critchley[9] has reported the neuropathological findings in lightning trauma as a ballooning of the myelin sheath of peripheral nerves and changes in the nerve fibres themselves, fragmentation and tortuosity of the axones, breaking down of the Schwann sheath and fusion of their nuclei, chromatolysis of the nerve cells of the cord and brain, and others.

The following case report is believed to represent a unique injury in that no trauma other than unilateral acoustic difficulty was manifested and there was almost no visible injury to the ear involved.

CASE REPORT.

C. S., Jr., a 30-year-old white male, first consulted me on July 23, 1948, having been exposed to a bolt of lightning some four hours previously. At the time of the accident, a 14-year-old boy standing just to the right of him was killed almost instantly. Upon recovering from the shock of the accident, the patient noted an immediate profound loss of hearing along with a roaring noise in the right ear. As far as he could determine, his hearing was undisturbed in the left ear. He recalled that there was a "sharp crack" as the lightning struck, but remembered no thunder.

Prior to this time he had had no difficulty with his ears, and his nose and throat history was noncontributory. An adenotonsillectomy had been performed 25 years previously. There was no family history of deafness. Examination of the ears, nose and throat revealed the right ear canal to contain a moderate amount of cerumen and when removed the drumhead was seen to be markedly injected, with an absence of the light cone, although the position of the drum appeared normal, and it was intact. The left ear canal was clean and the drumhead was in normal position with a light cone and there was a minimal amount of injection about the handle of the malleus. The remainder of the otolaryngological examination revealed no pathology.

The audiogram showed a nerve type hearing loss in the right ear with air conduction in the conversational tones showing 40 db, 50 db and 70 db loss, respectively (see Fig. 1). There was no hearing beyond the 4096

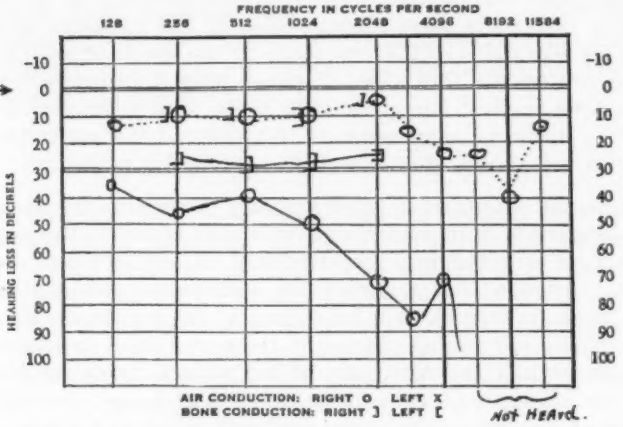

Fig. 1. Unbroken lines denote AC and BC loss immediately following injury. Broken line denotes AC and BC loss seven months later.

half tone frequency. The bone conduction showed a similar loss in the right ear. A sweep test of the left ear revealed all frequencies heard at a 15 db level. The Rinne test (256 fork) was bilaterally positive and the Weber test was not lateralized.

It was felt that the patient had suffered an electrical and concussion trauma to the ear mechanism, with some edema and irritation of the right drumhead. Accordingly, he was given Auralgan for the right ear twice daily and advised to return for re-examination.

The patient was next seen on July 27, 1948, at which time the drumhead showed minimal injection, and he stated that his hearing in the right ear was somewhat improved. The roaring had ceased the previous day. A second audiogram showed a 25 db, 15 db and 30 db loss, respec-

tively in the conversational tones for air conduction and the bone conduction showed a similar return toward normal. Hearing in the high tones was recovering but was still below the 30 db level. He was advised to discontinue all medication. When next seen on Aug. 21, 1948, he felt that there had been little improvement in hearing. Examination of the right drum revealed some retraction of the drumhead with dullness. Inflation of the ear revealed the Eustachian tube to be fully patent. The audiogram for air and bone conduction in the right ear was unchanged from the previous examination.

The patient returned March 11, 1949, for final examination. He stated that his hearing in both ears was normal and that his only difficulty was an occasional high pitched noise in the right ear. Both drums were in good position and of good color. The remainder of the otolaryngological examination was unchanged. His audiogram showed air conduction in the right ear to be within normal limits for frequencies through 2048. Higher frequencies showed 15 to 20 db loss in the right ear. Bone conduction was within normal limits in the right ear (see Fig. 1).

It is apparent from the final examination that the hearing disability was temporary and that recovery was almost complete in nine months. Since the original audiogram was essentially a nerve type loss, it would appear that either the cochlea or auditory nerve was affected. It was remotely possible that the hearing cortex may have been affected. The eardrum having returned to normal, and the frequencies through 2048 showing definite improvement in one week, would suggest only slight damage to the conduction mechanism. It was impossible to ascertain the pathology causing the moderate low tone and severe high tone loss after only a few weeks. The final audiogram indicates that the cochlea or auditory nerve (or possibly the hearing cortex) was involved in an edematous process, with or without hemorrhage. This pathology could be similar to the type seen in acoustic noise trauma, where the pathology is most marked in the higher frequencies located back of the round window. It is improbable that any electrical injury occurred to the cochlea, the auditory nerve or the cerebral cortex, in view of the complete recovery.

SUMMARY.

1. A number of bizarre lightning injuries, with special reference to trauma of the ears, are reviewed.

2. A case of uncomplicated, unilateral hearing loss following lightning trauma is presented.

3. An explanation is offered for the temporary hearing loss following lightning trauma without appreciable visible injury to the eardrum.

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TRANSITIONAL CELL CARCINOMA OF THE NASOPHARYNX IN A CHILD.

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A transitional cell carcinoma of the nasopharynx in a two-year-old child is an unusual tumor. Among 2,000 cases of neoplasia seen at the University of Michigan from 1895 to 1913, no case of carcinoma of the nasopharynx in a very young child is recorded,[1] and Pack and LeFevre[2] found only two nasopharyngeal epitheliomas from birth to four years of age out of a series of 16,565 malignant tumors seen at the Memorial Hospital in New York City. Friedman and Rubenfeld[3] also, in a smaller series of 3,161 neoplasms seen at Bellevue Hospital from 1925 to 1932, found only four carcinomas in children between one and five years of age. None, however, was located in the nasopharynx. Of the three tumors located in that region, the youngest patient was 16 years of age.

Besides being a rare type of tumor, its occurrence is unusual in the birth to five years of age group. Dargeon[4] reported seven cases of nasopharyngeal carcinoma from 1930 to 1937, the average age being seven years. The youngest patient he reported was four years old. New and Childrey[5] also found their youngest case to be four years old, in a series of 74 cases of malignancy of the palate and pharynx collected during the years 1917 to 1930. New and Hertz[6] state that the youngest patient they have seen was a three-year-old boy with a Grade IV squamous cell epithelioma who died within two months after the onset of symptoms.

The latter patient and the one reported in this paper appear to be among the youngest cases on record.

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CASE REPORT.

C. W., a 29 months old child, entered the Milwaukee Children's Hospital on Dec. 15, 1948. Five weeks before admission, she had been taken to another hospital with a sore throat and a draining ear. Her breath was noted to have a foul odor at that time. A "retrophayngeal abscess" was diagnosed and drained surgically, and a growth of "granulation tissue" was noted in the pharynx, but no biopsy was taken. She was given three weeks of penicillin therapy but did not improve and lost weight, so was taken home. She was kept at home for two weeks and then admitted to the Milwaukee Children's Hospital with the complaints of poor appetite and weight loss. The right eye had become "crossed" one week prior to admission and the foul breath had returned, although the ear no longer drained.

Her birth and developmental histories were normal, and the family history was noncontributory.

On examination, the child appeared acutely ill, dehydrated and weak. She held her mouth open constantly, and her breath was very foul. Palpation of the skin showed little subcutaneous fat and loss of normal turgor. There were several large cervical nodes on the left which were matted and tender, and she resisted anterior flexion of the neck because of pain. Examination of the eyes revealed a right internal strabismus but no pupillary changes.

The posterior nares were completely obstructed with thick mucus, and she, therefore, breathed through her mouth. The soft palate was pushed downward and forward by a rather firm mass which protruded into view posteriorly, and appeared dark brown, crusted and necrotic. It seemed a little fluctuant on the right to manual examination. There was marked surrounding edema and cellulitis with obstruction to nasal respiration.

The remainder of the examination and neurological was normal. The admission impression was a retropharyngeal abscess, but foreign body and neoplasm were to be ruled out.

Her fever on admission was 103° F., and the smear and culture of the nasopharynx revealed pus cells, staphylococci and H. influenzae, but no C. diphtheriae. She was, therefore, placed on parenteral penicillin and streptomycin and the dehydration was corrected by subcutaneous fluids. X-ray examination on the day after admission revealed normal sinuses. Lateral views, however, showed widening of the retropharyngeal and retronasal spaces. The retrolaryngeal space was normal. No air was noted in the posterior nasal passages, and there was no sign of pathology in the cervical spine.

After several days of therapy, the edema and cellulitis began to subside and another manual examination revealed only swollen, boggy, friable tissues in the nasopharynx, but no foreign body. A catheter could be passed completely through the right nostril into the posterior pharynx, but it met obstruction in the left nasopharynx.

After eight days of parenteral antibiotic therapy, the fever became normal, but there was only slight improvement in the breathing and obstructive signs. On the fifteenth hospital day the right pupil appeared dilated and reacted sluggishly to light. There was a ptosis of the right upper lid. Fundus examination showed no papilledema but did demon-

strate dilated, tortuous veins. The postnasal swelling seemed smaller, and the palatal cellulitis was less marked. On manual examination at this time a mass the size of a cherry could be felt on the left side of the nasopharynx in the region of the fossa of Rosenmüller.

Puncture incisions through both sides of the soft palate and into this tumor were performed on the fifteenth hospital day, after it was believed that the maximum benefit had been obtained from medical therapy, and secondary infection had been controlled. Incisions were also made into the posterior wall of the pharynx on both sides, but no pus was obtained. Re-X-ray after this procedure revealed the nasopharynx still occluded, but less swelling was present retropharyngeally. No bone erosion was noted at the base of the skull, but a repeat X-ray four days later suggested a questionable erosion of the anterior arch of the first cervical segment.

As no pus was obtained from the multiple incisions, a biopsy of the tumor mass behind the soft palate was done. The specimen consisted of a soft yellow piece of tumor tissue, 1 cm. by 0.6 cm. by 0.4 cm. It was irregular and partially necrotic.

On microscopic examination, the tumor was made up of undifferentiated cells, irregular in size and shape, and arranged in very poorly formed acini. The nuclei were extremely hyperchromatic and occupied almost all of each cell. Mitotic figures were infrequent and appeared somewhat irregular. Many dead cells were present, and a large amount of fragmented nuclear material could be seen throughout the sections. The superficial part of the tumor was entirely necrotic. A Gram stain revealed many monilia spores and mycelia in this superficial necrotic tissue. Dr. Gorton Ritchie's diagnosis was a transitional cell carcinoma of the pharynx.

Following this diagnosis, 11 deep X-ray treatments of 210 r. each were given to both the left and the right nasopharynx. This therapy was completed on Jan. 19, 1949, and she was then discharged home to be followed as an out-patient. She did not return to the hospital, however, and died at home on Jan. 24, 1949, 10 weeks after the onset of symptoms.

No necrospy was obtained.

Laboratory: The urine examination on admission was positive for acetone, diacetic acid and albumin. Microscopically, there were occasional R. B. C. and 15 W. B. C. The blood count, on admission, revealed 5,800 W. B. C. per cm., with 69 per cent leucocytes. There were 12 gm. of hemoglobin per 100 cc., and the sedimentation rate was 30 mm. in one hour. During the course of hospitalization the hemoglobin fell, and a transfusion of 200 cc. of whole blood was given. After this transfusion the blood count showed 4,750 W. B. C. per cm., with 39 per cent leucocytes and 15 gm. of hemoglobin per 100 cc. The blood culture on admission revealed no growth, and the Kline test was negative.

COMMENTS.

Next to the retinal glioma, the most common location of cancer of the head and neck in children is in the region of the nasopharynx, tonsil and soft palate.[4] "Ordinarily the tumor is

a single, sessile, firm, circumscribed and movable growth with a smooth, nonulcerated surface."[5] The size commonly varies from 1 to 14 cm.[5] In this case the tumor was about 1 or 2 cm. in diameter, but it was the surrounding reaction and secondary infection that caused many of the obstructing nasopharyngeal signs.

Such tumors may extend into the nasopharynx, obstruct the choanae and Eustachian tube, push the palate forward and partially fill the mouth.[5] Occasionally they may erode the cervical vertebrae, as was apparently occurring in this patient. Usually, the transitional cell-type tumor is not bulky but, due to its peculiar location, it may present nasal or nasopharyngeal obstruction and/or earache. According to Dargeon,[4] "Nasal obstruction which comes on after two years should not be ascribed to adenoids."

The lymphoepithelium as found over the tonsils, vault of the pharynx and nasal passages presents histological and clinical peculiarities, and tumors of the nasopharynx, therefore, are often very anaplastic and highly malignant lymphoepitheliomas or transitional cell carcinomas, Grade III or IV.[4,7]

Often persistent cervical adenopathy is the first sign noted, and extensive metastases may occur before the local lesion is found.[3] In any case, therefore, with persistent asymmetrical cervical adenopathy, neoplasia should be considered and adequate search for the primary lesion in the upper respiratory tract should be made.[4,5]

Mortality due to carcinoma in childhood is extremely high, and most cases of carcinoma of the nasopharynx terminate in death in from two months to three years.[9] For example, the average duration of life from the first symptoms to the time of death was 20 months in Dargeon's[4] series of seven cases. A short course of only 10 weeks was present in our case.

Figi,[9] however, is one of the few authors who has reported a squamous cell carcinoma of the nasopharynx in an 11-year-old boy which apparently responded to radium therapy; how-

ever, even if the tumor is apparently controlled locally, generalized metastases often occur, and death results. Often these nasopharyngeal tumors are highly radiosensitive, but are usually not locally accessible to surgery.[4]

Rector,[10] in discussing carcinoma in young persons, states that in Michigan, from 1938 to 1942, there were 546 deaths due to carcinoma in persons under 20 years of age. "None of the communicable diseases, measles, smallpox, scarlet fever, pertussis or rheumatic fever come close to causing the number of deaths in Michigan each year that carcinoma does."[10] Judging from these figures, it appears that carcinoma in childhood is a relatively important cause of death, and the mortality rate is extremely high.[8] Statistical improvement may occur when earlier recognition of the disease permits therapeutic measures to be initiated more promptly.[8] Such an early diagnosis is often delayed, as the age of the patient is commonly a misleading factor. If the not unusual occurrence of neoplasia in children is kept in mind, however, and microscopic examination of suspicious tissue is undertaken earlier, it is hoped these disappointing statistics may be greatly improved.

SUMMARY.

1. A transitional cell carcinoma of the nasopharynx in a two-year-old child is an unusual tumor. The record of such a case is reported.

2. Carcinoma of the nasopharynx may be found only after a persistent, asymmetric, asymptomatic cervical adenopathy is biopsied.

3. Suspicious, refractory tumor masses in the nasopharynx, with or without obstructive symptoms, earache, or persistent foul drainage, should be biopsied promptly as they may be malignant.

4. Transitional cell carcinoma may be highly malignant, but is often very radiosensitive.

5. The mortality of carcinoma in children is extremely high and is a relatively important cause of death.

It is hoped that earlier biopsy, diagnosis and treatment will improve these statistics.

I thank Dr. H. Miner for his cooperation in presenting this case.

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NEW METHOD OF THREADING AN ESOPHAGEAL STRICTURE.

HENRY J. RUBIN, M.D.,
Los Angeles, Calif.

Passage of a string through the esophagus is sometimes imperative to forestall gastrostomy. Occasionally this preliminary step in treatment is most difficult, and it may be impossible.

The following method was devised under a peculiar set of circumstances. A female infant was born with a stenosis, approximately 2.5 cm. long, in the thoracic esophagus. Esophagoscopy revealed an abrupt narrowing of the esophagus in the midthoracic region to a small, centrally situated orifice about 3 mm. in diameter. A No. 10 bougie passed through tightly. Very slow feeding with dilute formula was possible; and the father, a physician, refused to permit gastrostomy for institution of retrograde dilatation as long as the child maintained itself. The suggestion of occasional esophagoscopies with bougie dilatation was likewise rejected. The infant swallowed a weighted string readily, but it coiled proximally to the stenosis and was repeatedly vomited up after 24 to 36 hours. An attempt to pass a small stringed lead shot on a positor through an esophagoscope was abandoned, after one attempt, as unsafe. When the child was seven months old, the father insisted that something be done to facilitate feeding and permit the addition of solid foods.

The following successful method of passing a string was devised: a small piece of lead foil was pounded to less than half millimeter thickness, shaped around a needle, and cut to

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one-quarter inch length. No. 3 linen "cord," which is thinner than ordinary string but much stronger, was threaded through the small lead cylinder, and the metal was gently hammered

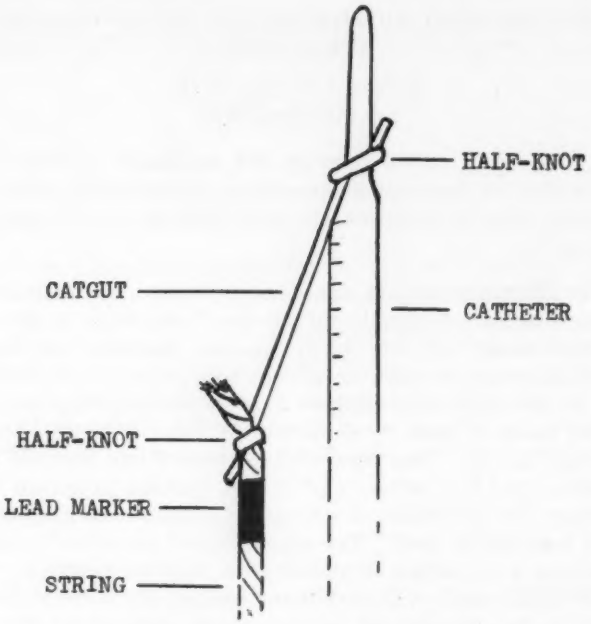

Fig. 1. Combined diameter of catheter and string is 3 mm.

until the malleable lead partially shredded the string and became so enmeshed in it as to be incapable of separation without the exertion of undue force. This was done to permit following the position of the string by fluoroscopy. The distal end of the string was cut so that only one-quarter inch protruded beyond the lead marker. With hemostats, a small piece of No. 000 catgut was tied tightly to this end of the string with only the first loop of a square knot, and the other end was tied in the same manner to the tip of a No. 6 woven

ureteral catheter. This catheter is sufficiently firm to be guided into and through the esophagus but buckles easily and is incapable of perforating the esophagus. The piece of cat-

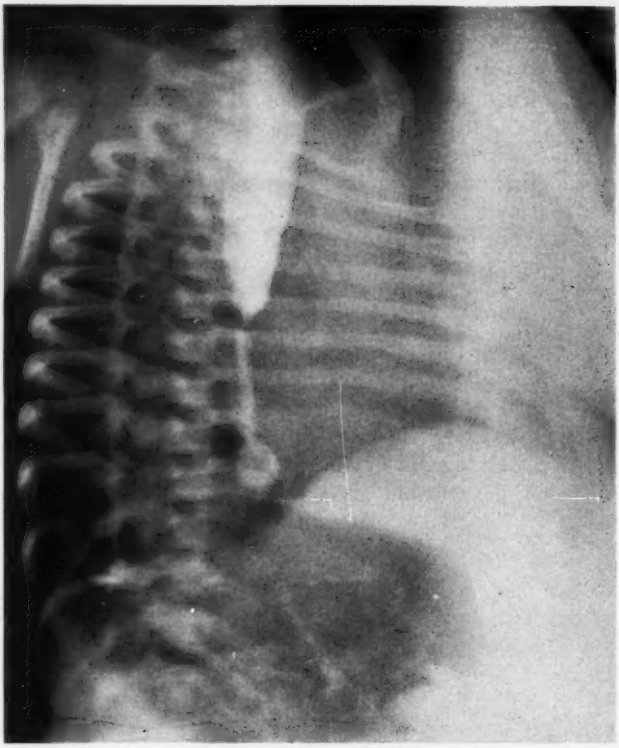

Fig. 2. Appearance of stricture through which string was passed.

gut was an inch long and connected the catheter and string with temporary security. The half-tied catgut knots were cut short.

Under fluoroscopic guidance, without anesthesia, and with suction for repeated pharyngeal aspiration, the catheter was passed into the esophagus. It came to an abrupt stop at the

level of the stenosis, and manipulation was begun to find the opening. After about five minutes of gentle pressure and twisting, it suddenly slipped through, and the lead marker was seen to enter the stomach. Further manipulation, turning and contact with stomach secretions served to loosen one of the half-tied knots, and within 10 minutes the catheter had slipped free and was withdrawn, leaving the string in the stomach. That the catheter actually lay free was easily determined, for the lead marker on the string no longer moved up toward the stricture as the catheter was started out. The course of the marker was then followed for several days as it moved through the intestinal tract. When the string had passed per rectum, dilatation with perforated metal olive-tipped bougies was begun and has continued successfully as an office procedure.

This method is suitable where a guide string in the esophagus is desired but cannot be passed, and gastrostomy is the alternative.

3875 Wilshire Boulevard.

THE RESEARCH STUDY CLUB.

The Nineteenth Annual Midwinter Postgraduate Clinical Convention in Ophthalmology and Otolaryngology will be held in Los Angeles, Jan. 15 to 27, inclusive. Given by the Research Study Club of Los Angeles, this event has come to be regarded as one of the outstanding meetings of the two specialties each year.

The featured speakers include Dr. Conrad Berens, Director of the Ophthalmological Foundation of New York and Chief of the New York Eye and Ear Infirmary, and his associate, Dr. Raymond E. Meek. The principal otolaryngologists will be Dr. Percy E. Ireland, Professor of Otolaryngology at the University of Toronto, and his assistant professor, Dr. Joseph A. Sullivan.

Special lectures of interest to otolaryngologists will be given by Dr. George C. Griffith and Dr. Samual L. Perzig, of the University of Southern California School of Medicine, Dr. Rea E. Ashley, of Stanford School of Medicine, J. C. Harding, D.D.S., of San Diego, "Nutrition for the Specialist," and Dr. Cecil H. Collins, of the University of Southern California College of Dentistry.

A special course in "Surgery of the Eye" will be given by Dr. Meyer Weiner, in which the various operations for correction of ptosis and epicanthus, along with sliding grafts for the reconstruction of the upper and lower lids, will be discussed. Other outstanding ophthalmologists on the program are: Phillips Thygeson, M.D., who will give The Third Doheny Lecture, Frederck C. Cordes, M.D., Professor of Ophthalmology of the University of California School of Medicine, and Archie E. Cruthirds, M.D., of Phoenix, Ariz.

Each applicant must be a member of his own local medical society in order to become eligible for attendance at the course. The fee (Clinical Course, $75.00; Special Course in Surgery of the Eye, $50.00) should be sent to Pierre Viole, M.D., Treasurer, 1930 Wilshire Boulevard, Los Angeles, Calif.

BOOK REVIEWS.

Recent Advances in Otolaryngology. Second Edition by R. Scott Stevenson, M.D., Ch.B.F.R.C.S., Surgeon, Metropolitan Ear, Nose and Throat Hospital; President, Section on Otology, Royal Society of Medicine. 106 text figures; eight plates; 395 pages, 1949. Philadelphia: The Blakiston Co. Price $6.00.

This is an excellent book, which, as the author states in his preface, is somewhere between a textbook and a year book. All the more recent work in otolaryngology is reported, with comments that are worthwhile.

There is an excellent bibliography at the end of each chapter and the illustrations borrowed from many sources are well reproduced.

The book is invaluable because it will bring the readers up to date in the newer work in the field and is delightful reading withal.

T. E. W.

Audiology. The Science of Hearing. A Developing Professional Specialty. By Norton Canfield, M.D., Associate Professor of Otolaryngology, Yale University School of Medicine; Board of Directors, Audiology Foundation; Board of Directors, American Hearing Society; Vice-President, International Audiology Conference; Consultant in Audiology, Veterans Administration; Consultant in Otolaryngology, Office of the Surgeon General, Department of the Army, Washington, D. C. With Foreword by Gunnar Holmgren, Professor of Otolaryngology, Stockholm, Sweden. Publication No. 51, American Lecture Series. 44 pages with bibliography. Springfield, Ill.: Charles C. Thomas, Publishers, 1949. Price $1.75.

This very short text, 38 pages, might be of help to the layman who wants to know what "audiology" means. It might be used as a starting point for interesting a civic-minded individual in raising funds for a hearing center. We wonder what other purpose the author had in mind when he wrote it.

T. E. W.

Acute Laryngotracheobronchitis. By A. Harry Neffson, M.D. 197 pages with index. New York (16), N. Y.: Grune & Stratton, Inc., 1949. Price $5.00.

This is an excellent book by an author whose vast experience qualifies him to write authoritatively. The whole subject of laryngotracheobronchitis is excellently and completely covered.

One may suppose that many will disagree with Dr. Neffson's enthusiasm for intubation; however, he states clearly the indications and contraindications for this procedure and emphasizes the need for expertness if intubation is to be contemplated.

The chapter on Tension Pneumothorax and Mediastinal Emphysema is very valuable and emphasizes a complication all too frequently undiagnosed.

The chapter on Treatment is very complete and is presented beautifully, clearly and concisely. Every phase is thoroughly covered.

This book should be a standard text in children's hospitals, in hospitals for infectious diseases and in postgraduate courses in otolaryngology.

T. E. W.

Clinical Audiology. By Maurice Saltzman, M.D., Assistant Professor of Otorhinology, Temple University School of Medicine; Associate Otolaryngologist, Mount Sinai Hospital, Philadelphia; Diplomate, American Board of Otolaryngology. Introduction by George M. Coates, M.D., Chief Editor, Archives of Otolaryngology, and Professor of Otolaryngology Emeritus, University of Pennsylvania. Foreword by Matthew S. Ersner, M.D., Professor and Head of the Department of Otorhinology and Rhinoplasty, Temple University School of Medicine. 195 pages with index and 82 illustrations. New York (16), N. Y.: Grune & Stratton, Inc., 381 Fourth Avenue, 1949.

This book, supposedly the latest word on Clinical Audiology, is a great disappointment. It is full of statements that lack proof; for instance, on page 25, "A sclerotic mastoid is a better conductor of sound than cancellous bone." Page 49, "Most often, however, it is the vascular twigs to the cochlea that have become involved in the arteriosclerotic process and as no collateral circulation is present in the basal turn, the sclerosing end of the arteriole must of necessity be followed by secondary nerve degeneration and a greater high frequency loss will be shown on the audiogram." Page 73, "Tinnitus aurium is a constant symptom of otosclerosis in the adult." Page 74, "In the prophylaxis and treatment of otosclerosis, dietary regulation and the administration of calcium, liver extract, insulin and vitamins are of considerable value according to Guggenheim."

From reading the chapter on Ménière's Syndrome and Symptomatic Ménière's Syndrome, one might assume that the diagnosis and treatment are simple. "As the etiology becomes evident, the therapeutic indications are clear-cut and the prognosis is predictable." O, that this were true!

It seems a pity that the author has not caught up with the latest available literature; his bibliography is most inadequate.

There are many audiograms, most of which could well have been omitted, and the chapters on Hearing Tests and Hearing Aids are deplorably inadequate. He hardly seems aware of the value of speech testing or how it is done. There seems to be little excuse for this publication.

T. E. W.

New Gould Medical Dictionary. A modern, comprehensive dictionary of terms used in all branches of medicine and allied sciences, including medical physics and chemistry, dentistry, pharmacy, nursing, veterinary medicine, biology and botany, as well as medicolegal terms; with illustrations and tables. Edited by Harold Wellington Jones, M.D.; Normand L. Hoerr, M.D.; Arthur Osol, Ph.D., with the cooperation of an editorial board and 80 contributors. Two hundred fifty-two illustrations on 45 plates, 129 in color. First edition. Philadelphia: The Blakiston Co. 1949.

This dictionary is comprised of a volume of material for which every field of medicine is represented in an up-to-date manner. Not only does the selection of material show clear, straight forward presentation but worthless entries which have cluttered previous dictionaries have been omitted. The pronunciation of words is indicated in simple form, and the standard for correctness is clearly shown. Proper names attached to structures or discoveries are usually to be found under a short biographical sketch. All tables are included in a section of their own at the back of the book instead of being scattered through it. The illustrations are likewise included in a single section, but near the center of the book. They represent 45 plates of well selected and clearly reproduced figures, for which references appear throughout the text. The type selected and the arrangement is an aid to the ease and amount of time used in finding a particular term.

This dictionary is one which will be most acceptable to all concerned with any aspect of medicine. W. P. C.

DECEMBER 1, 1949.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

As of December 1, 1949.

Aurex (Semi-Portable); Aurex Model C-B, Model C-A, Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Type K; Maico Atomeer.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St.. New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303.
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model; National Standard Model; National Star Model.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL; Paravox Model XT; Paravox Model XTS; Paravox Model Y (YM, YC and YC-7).

Manufacturer: Paraphone Hearing Aid, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear 45-CM; Radioear Model 45-M-magnetic air conduction receiver; Radioear Model 45-M-magnetic bone conduction receiver; Radioear Permo-Magnetic Uniphone; Radioear Permo-Magnetic Multipower.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic Hearing Aid Model 101; Silver Micronic Hearing Aid Models 202M and 202C.

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

Silvertone Model 103BM.

Distributor: Sears-Roebuck & Co., Chicago, Ill.

Sonotone Audicles No. 530, No. 531 and No. 533; Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.
Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 612; Telex Model 900; Telex Model 1020; Telex Model 1550; Telex Model 1700.
Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Orthotronic Model; Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.
Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Radionic Model A-2-A; Zenith Radionic Model A-3-A; Zenith Radionic Model B-3-A; Zenith Model 75; Zenith Miniature 75.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

TABLE HEARING AIDS.

Precision Table Hearing Aid.
Manufacturer: Precision Electronics Co., 850 West Oakdale Ave., Chicago 14, Ill.

Ravox.
Manufacturer: Zenith Radio Corp., 6001 W. Dickens Ave., Chicago, Ill.

Sonotone Professional Table Set Model 50. Report not yet published.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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Council Meeting, Hotel Statler, New York City, Jan. 6, 1950.
Combined Meeting, Southern and Middle Sections, Peabody Hotel, Memphis, Tenn., Jan. 16-17, 1950.
Western Section, Medical Society Bulding, Los Angeles, Calif., Jan. 21-22, 1950.

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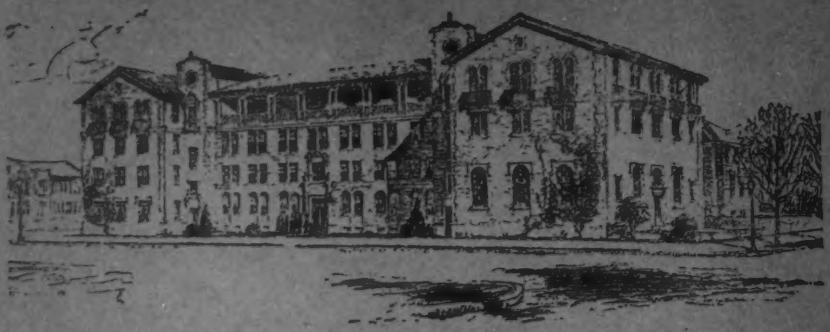

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CONTENTS

PSYCHOSOMATIC APHONIA AND EPHEMERAL ADDUCTOR PARALYSIS. Chevalier Jackson, M.D., Philadelphia, Pa. - - - - - - - 1287

THE FENESTRATION OPERATION FOR OTOSCLEROSIS. A SURVEY OF EXPERIENCE WITH 75 CASES TOGETHER WITH A STUDY OF THE EFFECT OF THE CONTRALATERAL EAR. De Graaf Woodman, M.D., New York, N. Y. - - - - - - - - - - - - - - - 1299

SURGICAL REPAIR OF CONGENITAL CHOANAL ATRESIA. J. Eastman Sheehan, M.D., and Wilson A. Swanker, M.D., New York, N. Y. 1320

DETECTION OF NOISE SUSCEPTIBLE EARS. D. E. Wheeler, Ph.D., Iowa City, Iowa - - - - - - - - - - - - - - - 1328

HEMANGIOMA OF THE LATERAL BAND OF THE PHARYNX AND NASOPHARYNX. CASE REPORT. W. W. Wilkerson, Jr., M.D., and Lee F. Cayce, M.D., Nashville, Tenn. - - - - - - - - - - 1339

SOLITARY NEUROFIBROMA OF THE LARYNX (REPORT OF TWO CASES). Gilbert E. Fisher, M.D., and John S. Odess, M.D., Birmingham, Ala. - - - - - - - - - - - - - - - - - - 1345

LIGHTNING AS A CAUSE OF HEARING LOSS. G. Brooks West, Jr., M.D., Cambridge, Md. - - - - - - - - - - - - - 1350

TRANSITIONAL CELL CARCINOMA OF THE NASOPHARYNX IN A CHILD. Bruce F. Grotts, M.D., Wilmette, Ill. - - - - - - - - - 1353

NEW METHOD OF THREADING AN ESOPHAGEAL STRICTURE. Henry J. Rubin, M.D., Los Angeles, Calif. - - - - - - - - - - 1361

THE RESEARCH STUDY CLUB. - - - - - - - - - - - - - 1365

BOOK REVIEWS. - - - - - - - - - - - - - - - - - - 1366

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION. - - - - - - - 1368

DIRECTORY OF OTOLARYNGOLOGICAL SOCIETIES. - - - - - - - 1371